COGNITIONS, CHRONICITY, DISTRESS AND DISABILITY IN TEMPOROMANDIBULAR DISORDER (IDIOPATHIC OROFACIAL PAIN)

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ABSTRACT

This thesis attempts to bring the understanding of orofacial pain, and temporomandibular disorder (TMD) in particular, into line with that of other chronic pain conditions, in order that the principles of psychological pain control through education and self-management, apparently effective in other conditions, may be specifically adapted to facial pain.

Reviews of the literature on coping, beliefs, depression, and anxiety, establish the current status of theoretically derived measurement instruments, their use in chronic pain patient groups, and the findings therefrom. Further reviews of psychological aspects of dental pain and primary headache present these as acute and chronic pain conditions for comparison.

The first study, assessing disability by means of the Oral Health Impact Profile (OHIP), found no significant relationships between OHIP factors and clinical signs. However, all OHIP factors, except for the functional and physical subscales, correlated significantly with pain intensity, suggesting that psychosocial rather than physical aspects of disability relate to pain report. Anxiety, measured by the Hospital Anxiety and Depression scale (HAD), was associated with pain intensity (McGill Pain Questionnaire, MPQ), catastrophising (Coping Strategies Questionnaire, CSQ), and with 'psychological' pain beliefs (Pain Beliefs Questionnaire, PBQ). In addition, anxiety appeared to be related to perceived problems with speech (OHIP). Depressive symptoms (HAD) were associated with 'passive' coping strategies, notably catastrophising, and with emphasis on the impact on tasting and digesting food (OHIP).

The second study sought to evaluate how pain symptoms of different character and intensity (MPQ) might influence cognitions relating to cause, timeline, consequence, and control/cure (Illness Perceptions Questionnaire, IPQ). Greater intensity of 'constant' pain contributed to greater advocation of physiological cause, and greater perceived consequence of pain on psychosocial functioning, whilst a longer period in full-time education appeared to increase endorsement of psychological causes, and to beneficially influence judgments of timeline and consequence. Facial pain, though reportedly less painful, of lesser consequence, and more controllable than headache, was also found to be less responsive to treatment, as demonstrated over six months following hospital specialist consultation. The contribution of perceived permanence and consequence (IPQ), and of catastrophising (CSQ), to continuing pain, disability, and distress indicates that these factors were suitable targets for a psycho-educational approach aimed specifically at ameliorating pain beliefs and coping strategies.

In the final study, a self-management programme for TMD was developed from the findings of the earlier studies, and successfully piloted in a small sample of patients, demonstrating the acceptability of the programme, indicating a need for a full-scale trial.

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Chapter 1. INTRODUCTION

1.I. AIMS OF THE THESIS

The orofacial region is affected by a number of chronic pain symptom complexes of unknown cause, of which temporomandibular disorder (TMD), is the most prevalent and therefore the greatest drain on health service resources (Bonica, 1980). This condition is of particular interest also, in that it generally affects young and otherwise healthy individuals.

Much research has been conducted on psychological aspects of chronic pain syndromes, yet orofacial pain has tended to be seen as a special case and managed by orofacial specialists. This thesis aims to bring the understanding of psychological factors in orofacial pain, and TMD in particular, into line with that in other chronic pain conditions, in order that the principles of psychological pain control through education and self-management, apparently effective in other conditions (Jensen et al, 1999), may be specifically adapted to facial pain.

The thesis will review the literature on three pain conditions for the purpose of comparison: temporomandibular disorder, acute dental pain, and chronic primary headache. Reviews will then be presented of studies of cognitive psychological factors in various chronic pain groups, including coping strategies; pain beliefs, depression, and anxiety, in order to determine how these factors may best be studied in temporomandibular disorder and the comparative groups.

Studies will then be described, which investigate cognitive factors relating to pain, disability and distress in temporomandibular disorder. Comparisons will be made with an acute post-surgical pain group (third molar removal) and a chronic pain

group in which psychological factors are more readily acknowledged (benign headache), with the intention of establishing what, if anything, is special about facial pain.

The ultimate goal is an intervention specifically aimed at ameliorating cognitions and behaviours in TMD, in order to reduce the associated pain, disability and distress.

1.II. DEFINING PAIN

Pain has been defined as *"An unpleasant sensation and emotional experience which is associated with actual or potential tissue damage or is described in terms of such damage"* (Merskey et al, 1979). Pain is not a simple sensation like touch and taste, but rather a complex perception, involving higher processing of peripheral nerve signals in the context of other factors, such as previous experience and current mood. In other words, pain is subject to psychological control. The Gate Control Theory (Melzack & Wall, 1965), which incorporates psychological mechanisms, remains the major working theory about pain despite the lack of neuro-histological evidence for some of its components. Patrick Wall eloquently describes several anecdotes illustrating the disjunction of pain and injury and the influence of private and public factors on pain (Wall, 1999: Ch. 1).

Pain of at least six months' duration is considered 'chronic'. Whereas acute pain may serve a useful biological function in alerting the body to tissue damage in order to seek protection from further damage, chronic pain appears to have no such purpose and, rather, may inhibit the individual's capacity to function and to enjoy life. Chronic pain is often elusive to diagnosis ('idiopathic') and recalcitrant to treatment.

Chapter 2. TEMPOROMANDIBULAR DISORDERS

2.I. CLASSIFICATION

Current diagnostic terms include tension headache, migraine, neckache, temporomandibular disorders (TMD, temporomandibular joint pain dysfunction syndrome - TMPDS, facial arthromyalgia) and atypical facial pain. These pains appear to arise from blood vessels, muscles and joint capsules rather than sensory nerve branches as in trigeminal neuralgia. Considering symptoms, there are four recognisable complexes which may, however, coexist: temporomandibular disorder (TMD, TMPDS, facial arthromyalgia, myofascial face pain); atypical facial pain (atypical facial neuralgia); atypical odontalgia (phantom tooth pain); and oral dysaesthesia (burning mouth syndrome, glossodynia, glossopyrosis).

Temporomandibular Disorders (TMD) comprise the most common non-infective pain condition of the orofacial region (Lipton et al, 1993). TMD is clinically characterised by pain within and around the temporomandibular joint(s) and adjacent muscles of mastication, clicking of the joints, sometimes limitation of mouth opening and rarely locking of the joint in opening or closing (Truelove et al, 1992).

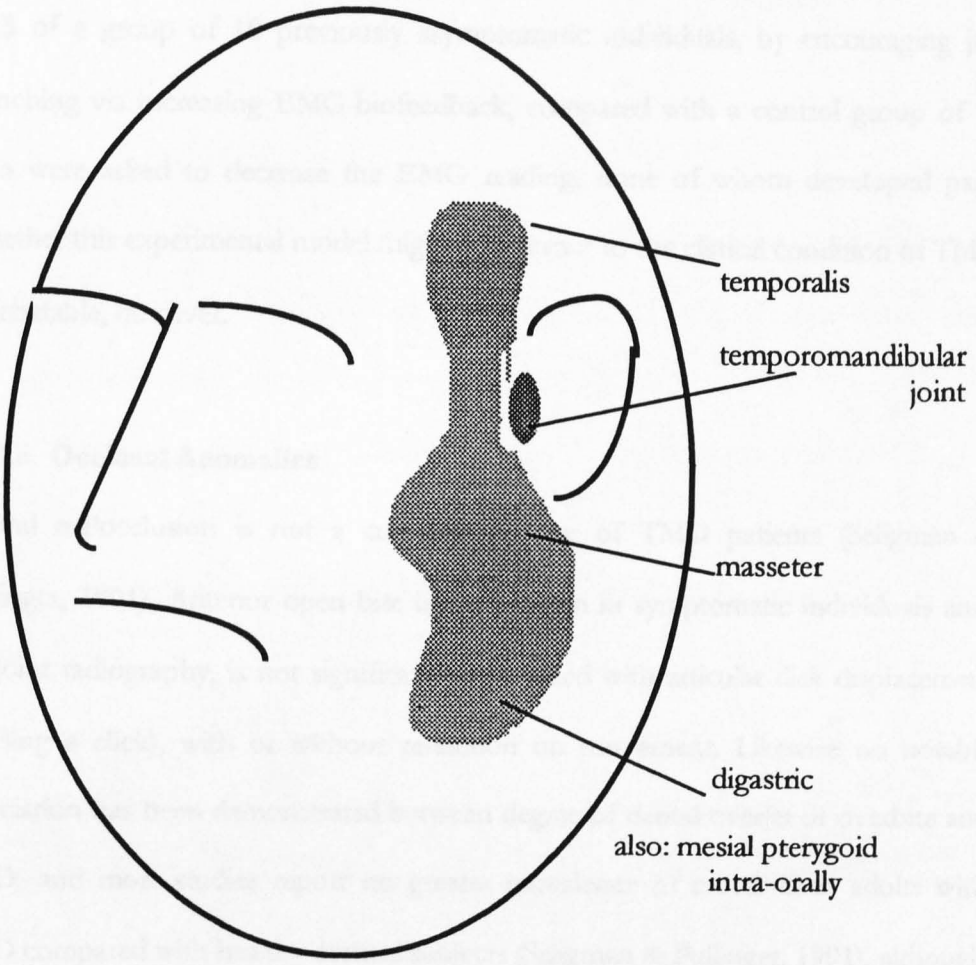
There is considerable disagreement between authorities over the precise clinical features, and hence the diagnostic criteria for TMD. Indeed, the terminology used to describe this symptom group varies widely. As a consequence of these problems, the literature is often confusing and difficult to interpret.

2.II. EPIDEMIOLOGY

In general, up to 67% of groups of children, young adults, and semi-selected and randomly selected patients have experienced ay least one painful symptom of the temporomandibular joint (TMJ) and associated structures, the majority of complaints being minor and transient (Porter, 1996). Clinical examination can detect associated signs in up to 69% of examined persons, and these signs can often be found in asymptomatic individuals (Schiffman et al, 90). Studies of US households suggest that as many as 8% of interviewed persons had had TMJ or facial pain more than once in the previous 6 months (Lipton et al, 1993) but the intensity of the associated pain may vary considerably with time and may be recalled inaccurately.

Symptoms and signs of TMD can arise in children as young as 3 years of age (Mintz, 1993). The prevalence of TMD has been suggested to increase towards middle age and then gradually fall but this is not a consistent finding (Locker & Slade, 1988). Although a female predisposition has long been suggested, the frequency of symptoms and signs may be similar in both genders (Salonen et al, 1990). However, females, particularly in the third and fourth decades, may have more severe clinical upset (headache, joint and muscle tenderness, and joint clicking; Ohna et al, 1988), may recognise painful symptoms better than do males, and may more readily seek professional therapy than do affected males (Centore et al, 1989). Despite the apparently high frequency of symptoms and signs of TMD, perhaps only 2-7% of sufferers actually seek, and perhaps warrant, treatment (De Kanter et al, 1992).

Figure 2.I. Diagram to illustrate the pain distribution in TMD



2.III. AETIOLOGY – LOCAL FACTORS

2.III.i. Parafunctional Habits

Parafunctional habits such as biting foreign objects, pressing the tongue against the teeth, lip biting, clenching and grinding, may have a variable and possibly minor association with TMD (Schiffman et al, 1992). The assessment of parafunctional habits may be complicated and influenced by the self-reporting of patients and/or the abilities of the attending clinician, in terms of accuracy of recall and response to loaded questions (Marbach et al, 1990; Marbach, 1992).

Glaros et al (2000) appeared to induce TMD-like symptoms (myalgia and arthralgia) in 3 of a group of 10 previously asymptomatic individuals, by encouraging jaw clenching via increasing EMG-biofeedback, compared with a control group of 10 who were asked to decrease the EMG reading, none of whom developed pain. Whether this experimental model might be relevant to the clinical condition of TMD is debatable, however.

2.III.ii. **Occlusal Anomalies**

Dental malocclusion is not a common feature of TMD patients (Seligman & Pullinger, 1991). Anterior open bite is uncommon in symptomatic individuals and, on joint radiography, is not significantly associated with articular disk displacement (causing a click), with or without reduction on movement. Likewise no notable association has been demonstrated between degree of dental overjet or overbite and TMD, and most studies report no greater prevalence of crossbite in adults with TMD compared with healthy control subjects (Seligman & Pullinger, 1991), although an association between contralateral crossbite and reducing disk displacement may exist (Robergs et al, 1987).

Some, but not all, studies of TMD patients have suggested an association between molar loss and pain, clicking and progression to locking, but there is little correlation between loss of molar support and TMD symptoms in randomly selected individuals (Pullinger & Seligman, 1991). Incidentally, there is no evidence for any association with dental attrition (Seligman et al, 1988).

Some studies have suggested that an asymmetric retruded contact position can cause abnormal joint sounds and masticatory muscle tenderness, but there is not always a

significantly increased frequency of asymmetric retruded contact position in TMD groups (Pullinger et al, 1988). However, an abnormal retruded contact position may be a feature in some patients with uncommon, specific joint derangements (Seligman & Pullinger, 1989).

There may be a higher frequency and severity of TMD in patients with restored dentitions compared with those with intact dentitions, but the precise contribution to TMD aetiology is unclear (Kamper & Hahnerz, 1991).

Skeletal factors and orthodontic treatment probably play little role in the aetiology of TMD (Sadowsky, 1992). Similarly the long-term effects of orthognathic surgery on TMD are not clear (White & Dolwick, 1992).

2.III.iii. **Trauma**

Traumatic injuries from eating, wide opening, and dental treatment have all been cited as possible aetiological factors but there is little objective evidence to support this.

Previous head and neck injury may be a feature of patients with TMD (Pullinger & Seligman, 1991); such trauma may precipitate pain and may underlie the arthroscopic findings such as synovitis, fibrillar organisation, and adhesions (Harkins & Morteney, 1985). It is unclear whether a specific injury gives rise to a particular increased frequency or type of TMD but, since only mild to moderate dysfunction occurs in patients with previous mandible condyle fracture (Dahlstrom et al, 1989), it seems unlikely that this local trauma is of aetiological significance. There are inadequate data to support whiplash injury as a likely precipitant of TMD (Truelove & Blasberg, 1993).

Whilst there may be an increased frequency of generalized joint hypermobility in some TMD groups (Buckingham et al, 1991), and children with joint hypermobility may have an increased liability to TMJ pain (Adair & Hecht, 1993), it seems unlikely that joint laxity is a significant aetiological factor in TMD.

2.IV. AETIOLOGY – PSYCHOLOGICAL FACTORS

2.IV.i. Life Events

Stressful life events may be more frequently reported in groups of TMD patients than in non-affected control groups (Fearon & Serwallen, 1983; Stein et al, 1982). However, this association exists only for patients with muscle-related symptoms (Schiffman et al, 1992), and it is worthy of note that bruxism (tooth-grinding) and/or myofascial pain may themselves adversely affect quality of life (Bush & Harkins, 1995). An increased prevalence of post-traumatic stress disorder in TMD patients has been suggested but remains unconfirmed (Aghabeigi et al, 1992).

2.IV.ii. Psychiatric Illness

Whilst there is equivocal evidence for an association between psychiatric illness and TMD, many dental surgeons are of the belief that the two are linked (Glaros et al, 1994). Anxiety (Speculand et al, 1983), other affective disorders (particularly depression; Feinmann, 1985), somatoform disorders (Beck & Dimitroff, 1990), and personality disorders (Schulfe et al, 1993; Aghabeigi et al, 1992) may be more frequent in groups of TMD patients than in control groups. Forty percent of one US patient study group satisfied the diagnostic criteria for at least one personality disorder, the most common being obsessive-compulsive disorder (Kinney et al,

1992). Minnesota Multiphasic Personality Inventory (MMPI) scores from another group of US TMD patients revealed significantly higher levels of hypochondriasis, depression, hysteria, psychopathic deviation, paranoia, schizophrenia, and social introversion, than did controls (Bianchi et al, 1992).

All these findings are based on correlational studies and can therefore not be considered as evidence for psychiatric *cause* of TMD.

2.V. DIAGNOSIS AND ASSESSMENT

2.V.i. Clinical Signs

Traditional signs of TMD have included temporomandibular joint sounds, mandibular mobility and pain elicited on palpation of the joint and associated musculature. The prevalence of joint sounds (Pollmann, 1993) and the wide range of mandibular mobility (Szentpetery, 1993) in healthy populations, together with the subjectivity of mobility assessment and pain report on palpation (Ohrbach & Dworkin, 1998), mean that these signs must be interpreted with considerable caution. Also, joint sounds may remain unchanged despite perceived improvement of TMD after treatment (Resine & Weber, 1989; Ohrbach & Dworkin, 1998).

Assessment of joint sounds is not a reliable diagnostic technique (Mohl & Dixon, 1994). Thermography (Graff & Sickles, 1993), vibration analysis (Ishijaki et al, 1993), and jaw tracking devices (Mohl et al, 1990) are not of proven benefit in diagnosis. Likewise analysis of the electromyographic activity of masticatory muscles is rarely helpful in the diagnosis or monitoring of TMD.

There is growing support for a distinction between muscle-related ('myogenous') TMD and joint-related ('arthrogenous') TMD (Schiffman et al, 1992; Kight et al,

1999, Epker et al, 1999), and, hence, pain on palpation may be the most useful clinical measure. The term 'facial arthromyalgia' has been suggested in preference to 'temporomandibular disorder' since it refers purely to pain in the joint and/or muscles, ignores clicking and stiffness of the joints, and evokes comparison with other myofascial pain conditions (Cimino et al, 1998). TMD remains the internationally recognized term, however, and will therefore be used here.

2.V.ii. **Disability**

Following on from work with other chronic pain groups, attempts have been made to quantify the disability of TMD in terms of functional, psychological and social impact. One study (Reisine & Weber, 1989) evaluated patients with seven subscales of the Sickness Impact Profile (Bergner & Bobbitt, 1981) to establish problems with sleep, rest and concentration. Another study (Dao et al, 1994) used 5-point category scales to assess quality of life in bruxers and masticatory myofascial pain patients, whilst a third (Bush & Harkins, 1995) found the factor structure of the Pain Disability Index (PDI) in orofacial pain patients to mirror that for other chronic pain groups. The Medical Outcomes Study (MOS-17) was used to show improvement in social function and bodily pain after physical therapy (Di Fabio, 1998).

One measure developed specifically for the assessment of health outcomes of oral conditions is the Oral Health Impact Profile (OHIP, Slade & Spencer, 1994). This scale was developed in Australia using methods similar to those employed for the widely used generic Sickness Impact Profile, and has been used on a sample of older adults (Locker & Slade, 1993) as well as on a sample of mixed craniofacial pain patients (Murray et al, 1996), both in Canada.

2.V.iii. **Psychological Distress**

Table 2.I. summarises a germane sample of studies concerning mood in facial pain patients. Correlational studies of facial pain patients have found greater prevalence of depressive symptoms than in the normal population (33% in TMD, Kinney et al, 1992). Chronic facial pain patients exhibit reduced tyramine conjugation, a marker for depression, which may indicate a common pathogenesis for pain and depression (Aghabeigi et al, 1993). There may also be a history of post-traumatic stress disorder (Aghabeigi et al, 1992). Approximately 50% of acute TMD patients exhibit anxiety disorders (Gatchel et al, 1996) compared with 10% of chronic sufferers, indicating that anxiety may be an early feature. Psychopathology may also discriminate myogenous and arthrogenous groups of patients (Kight et al, 1999).

Table 2.I. Studies of mood in TMD patients:

Author	Sample	Design	Diagnosis		Results
			Physical	Other	
Vimpari et al (1995)	780 community subjects	correlational	TMJ sounds, limited opening, self-reported pain	Zung's Self-rating Depression Scale	subjective and objective symptoms of TMD more common in depressed subjects
Zautra et al (1995)	110 female myofascial face pain S's (private specialist)	longitudinal	tenderness in muscle(s) of mastication; TMJ sounds or limited opening	2 in-person and 10 telephone interviews over 12 months	pain and distress stable across months; 'trait' components correlated; 'state' components correlated; increases in monthly reports of pain preceded by elevated psychological distress in previous month.
Korszun et al (1996)	72 chronic facial pain patients	correlational	pain diagnosis from history, clinical and radiographic examination	DSM-IV (single psychiatrist)	53% major / minor depression 22% depressive symptoms
Gatchel et al (1996)	50 chronic and 51 acute TMD S's (> or < 6 months)	correlational	RDC/TMD	DSM-III-R	both groups showed greater-than-normal lifetime and current prevalences of psychopathology; acute group showed higher rates of anxiety disorders and lower rates of affective disorders than chronic group.
Kight et al (1999)	227 acute and chronic TMD S's	correlational	RDC/TMD	SCID	mood disorder, personality disorder and muscle disorder significantly related ($p<0.01$)

NB. See Key to Abbreviations (p. 261)

2.V.iv. **Predicting Chronicity**

A model for predicting chronic TMD has been arrived at (Epker et al, 1999). This study claims a 91% prediction of cases of acute sufferers going on to develop chronic symptoms from two initial variables: presence of myofascial pain on palpation (as opposed to other signs), and high 'characteristic pain intensity'. The latter is a combined pain measure comprising the mean of: current pain (0-10); worst pain over 3 months (0-10); and mean pain over 3 months (0-10); multiplied by 100. Problems with the study include the questionable validity of this pain measure, especially when pain for 3 months might already be considered chronic, and the inclusion criteria for 'acute' patients. Epker et al defined acute patients as being those who either had never previously sought treatment, or who had sought treatment within the previous six months and no more. This reliance on initial treatment-seeking is somewhat perplexing, considering the reliability of patients' memory of onset of symptoms reported elsewhere (Raphael and Marbach, 1997). Patients were considered to have developed chronic TMD if they continued to have pain, beyond a negligible cut-off score of 15, at follow-up, six months after the initial assessment. The study lends further support to the distinction between muscle pain and other supposed signs of TMD.

The Multidimensional Pain Inventory (MPI, Kerns et al, 1985) has also been cited as a means of predicting chronicity in TMD (Epker & Gatchel, 2000). Acute TMD patients with a 'dysfunctional' or 'interpersonally distressed' profile on the MPI were more likely to continue to complain of pain at 6 months than were those without such a profile. The same criteria were employed for acute and chronic TMD as in the previous study (Epker et al, 1999). The authors conclude that: "additional

implications for type of MPI coping style on medical/dental and psychosocial interventions in patients continue to be important topics for research" (p.75). Coping style is therefore to be addressed in this thesis.

2.VI. TREATMENT

2.VI.i. Homecare Practices

Homecare practices are favoured by most clinicians. However, there is a lack of data conceming their precise clinical benefit in the treatment of TMD. Commonly used homecare procedures may include avoidance of excess chewing, change to a soft-consistency diet, limited talking, avoidance of wide yawning, use of physical therapy such as local application of ice for acute pain or heat for low-grade chronic pain (Selby, 1985; Clarke et al, 1990), muscle massage, hot showers, saunas and steam baths (Greene, 1991).

Passive or active jaw exercises have been recommended for joint clicking, restricted opening, irregular mandibular movements, muscle incoordination, and recurrent anterior dislocation of the condyle (Selby, 1985). The results of one study suggested that exercises and physiotherapy successfully reduced pain and improved jaw opening in 53% of patients with reciprocal TMJ clicking (Kirk & Calabrese, 1989).

2.VI.ii. Analgesia

Pain, and possibly inflammation, may be controlled by non-steroidal anti-inflammatory drugs (NSAIDs; Clarke et al, 1990; Greene, 1991) but there do not appear to be any documented trials for their efficacy in TMD. A recent study found a combination of NSAIDs and mouth-opening exercises over four weeks to produce

objective improvement in 60% of patients with disk displacement without reduction compared with 33% improvement in a no-treatment control group (Yuasa et al, 2001)

2.VI.iii. **Splint Therapy**

A variety of occlusal splint designs has been reported to be of value in the management of TMD. Hard acrylic splints may be effective in reducing muscle and joint pain in up to 87% of studied patients (Greene & Laskin, 1972) but are unlikely to reduce joint clicking and limited opening (Okeson et al, 1982). Nocturnal splint therapy may be effective in reducing the symptoms of myogenous pain but arthrogenous pain requires continuous splint use, at least in the short term (Wilkinson et al, 1992). In the long term, patients often have a return of painful symptoms after cessation of splint use (Sheikholeslam et al, 1986). Flat plane splints may rapidly reduce nocturnal bruxism (Solberg et al, 1975) and sometimes, but not always, effect a decrease in maximum masticatory muscle activity (Carr et al, 1991; Shen & Yun, 1991).

Despite the lack of well designed studies, current evidence suggests that anterior repositioning splints are more effective then flat plane splints in eliminating reciprocal clicking and TMJ tenderness, and may also sometimes be more effective in reducing muscle tenderness (Lundh et al, 1988; Tallents et al, 1990). However, clicking often returns and anterior repositioning splints are only likely to obtain long-term recapture of the disk in one third of all treated patients (Lundh et al, 1988; Greene & Laskin, 1988).

Only about 50% of clicking, painful joints may be suitable for repositioning therapy, hence clinical benefit is unlikely in all treated cases (Okeson et al, 1988), and the success of therapy may not be dependent on the precise malposition of the articular disk (Tallents et al, 1986). A further disadvantage of the anterior repositioning splint is that, after the mandible has been maintained in an anterior position, it must be stepped back to the original occlusal position; at this time a posterior open bite may develop because the condyle has not completely returned to its original position within the fossa, necessitating later orthodontic therapy or complex occlusal adjustment (Okeson, 1991).

The reduction in pain may (theoretically at least) be due to the forward positioning of the disk, allowing the retrodiskal tissues space to repair, but, if the retrodiskal tissues have not fully repaired when the condyle is returned to the fossa, there will be further inflammation (Okeson, 1991).

Soft splints may lessen TMD-related headache and clicking (Quayle et al, 1990) but there effect is not always significant, particularly in the long term, and they can cause a worsening of symptoms in up to 26% of patients (Okeson, 1987).

Available limited data suggest that pivotal splints may be of benefit in reducing the pain of TMD but additional data are required (Lous, 1978). Buccal separators are not effective in TMD (Abraham et al, 1992).

2.VI.iv. **Occlusal Readjustment**

As noted above, there is only equivocal evidence for a role for occlusion in the aetiology of TMD (Tsukiyama et al, 2001). Nevertheless, occlusal readjustment continues to be cited as a useful therapeutic technique, particularly when myalgia is a major symptom (Long, 1992). Occlusal readjustment involves repositioning the

mandible in a centric position by prosthodontic or orthodontic means and/or occlusal equilibration. Most studies have only assessed short-term outcome (Lundh et al, 1988) but one study, of a small number of patients with TMD associated with abnormal condyle-disk relationship, reported significant reduction in painful symptoms and locking for up to 3 years after occlusal correction by prosthodontic and/or orthodontic therapy (Lundh & Westesson, 1989). Patients with asymptomatic clicking of the TMJ may benefit from replacement of any lost posterior teeth (Sorgi et al, 1992), but this assumes, probably incorrectly, that loss of posterior teeth causes TMJ clicking and that patients with asymptomatic TMJ sounds require treatment.

A systematic review of randomized controlled trials (RCT's) of occlusal treatment studies was undertaken by Forssell et al (1999). Splint therapy was found superior to 3 control treatments, and comparable to 12, and superior or comparable to 4 passive controls. Occlusal adjustment was found comparable to 2 and inferior to one control treatment and comparable to passive control in one study.

2.VI.v. **Surgery**

There is no evidence for the long-term efficacy of surgical treatment in controlled studies of TMD, although improved functioning has been reported in an 8 year follow-up of surgery in 70 patients (Peltola et al, 2000). US guidelines thoroughly discourage irreversible intervention (NIH, 1996).

2.VI.vi. **Psychotropic Medication**

Referral for psychiatric assessment and suitable therapy is an important component of TMD management (Feinmann & Harris, 1984,i).

A number of psychotropic agents have been suggested to be of value, of which dothiepin hydrochloride has probably received the greatest attention. Dothiepin hydrochloride in daily doses of between 25 and 225 mg can significantly reduce the painful symptoms of TMD after 9 weeks of therapy, but it may not reduce any associated depressive symptoms in that time (Feinmann & Harris, 1984,ii). A study of Australian patients found that both occlusal splint therapy and dothiepin therapy were required to reduce TMD symptoms in those patients with depression, whilst non-depressed patients responded poorly to splint therapy alone and had an intermediate response to dothiepin alone (Tversky et al, 1991).

Other suggested agents include amitriptyline (Gessel, 1975) and trifluoperzine hydrochloride in addition to dothiepin; fluphenazine with nortiptyline may be useful noctumally, and flupenthixol 0.5-1.5 mg twice daily may be of benefit in resistant cases (Feinmann & Harris, 1984,ii). Diazepam (2-5 mg up to three times daily) may reduce the pain of TMD (Jagger, 1973), and clonazepam has been found to reduce painful TMJ, head\and neck symptoms at nocturnal doses of 0.25-1 mg (Harkins et al, 1991). Alprazolam, a much more potent anxiolytic than diazepam, has been found to increase mandibular movement and decrease local pain and muscle tenderness, but does not significantly reduce joint sounds. Therapy with a flat plane occlusal splint may be as effective as alprazolam, and combined drug and splint therapy does not significantly positively influence clinical outcome (Nemcorsky et al, 1992).

2.VI.vii. **Complementary Therapies**

Acupuncture therapy may provide some reduction in local pain and tendemess, but this benefit lasts less than six months (List et al, 1993). Mandibular manipulations of various types have been suggested but consistent supportive data are required to determine long-term benefit and whether additional treatment is warranted.

Ultrasound may benefit some individuals with TMD but there appear to be few, if any, controlled studies (Mohl et al, 1990). Similarly, the benefit from photophoresis is unknown.

Studies of transcutaneous electrical nerve stimulation (TENS) have often lacked suitable control groups, have involved small samples, and have used inappropriate methods of assessment (Clark et al, 1987). Electromyography (EMG) has been extensively used in conjunction with relaxation and biofeedback therapy, and current data suggest that, whilst EMG biofeedback may provide some control of nocturnal bruxism, the benefit seems to be short lasting. Diurnal biofeedback relaxation is ineffective in reducing nocturnal bruxism (Pierce & Gale, 1988).

2.VII. SUMMARY

In summary, TMD is a common condition, particularly in young women, and involves pain in the temporomandibular joint and/or associated masticatory muscles. Diagnosis of joint disorder may be aided by imaging techniques such as MRI but diagnosis of muscle disorder relies on patient self-report. Assessment of distress and disability may be a useful adjunct to diagnosis. Manifold postulated aetiological factors are supported by anecdotal, correlational, or retrospective association and

remain unproven. Hence a disparate battery of therapeutic modalities has been advanced and a dearth of methodologically sound research has found none superior.

The next chapters will consider two other pain conditions, with which TMD is to be compared: acute dental pain and chronic primary headache.

Chapter 3. ACUTE DENTAL PAIN

3.I. INTRODUCTION

In the field of dentistry, the study of pain and its psychological manipulation has largely involved acute pain. As has been noted above, acute pain is generally considered useful and chronic pain a nuisance. In dentistry, fear of pain at the hands of the dentist keeps people away, despite the advances in pain control brought about by modern local anaesthetics. Acute dental pain might also therefore be considered a nuisance. Efforts to manage pain-related dental anxiety have shown some degree of success and might be adaptable to the problem of chronic TMD pain. This chapter reviews the relations between report of dental pain and psychological factors and interventions. Post-surgical pain is also included as this will provide a suitable acute pain model for comparison with TMD, in terms of pain site and patient age range.

The report of pain, during or following dental procedures, is largely governed by anxiety (Vassend, 1992). Anxiety about dentistry most commonly involves fear of pain (McNeil & Berryman, 1989). Lindsay et al (1987) found 84% of adults to expect sudden discomfort on occasion at the dentist's. This expectation of pain may be explained by the finding that individuals believe expected pain to be less intense than unexpected pain (Madland, 1988). Hence, anxiety is seen as a positive strategy in reducing pain intensity, in the event of being hurt suddenly.

3.II. CLASSIFICATION

Acute pain in dentistry may involve toothache, dental sensitivity, pain during treatment and post-operative pain. In psychological studies 'dental pain' refers to iatrogenic pain at the hands of a dentist. The experience of non-iatrogenic toothache is not generally considered to be stress-related (Sternbach, 1986), but is decidedly disruptive of daily life (Locker & Grushka, 1987). Yet psychological factors are involved in non-iatrogenic and post-operative dental pains, for example in terms of placebo effects with analgesia.

3.II.i. Toothache

Toothache does not affect perception of other pains. No differences were found in measures of thermal pain perception and forearm ischaemic pain between a group of 10 subjects with painful toothache and 7 age- and sex-matched pain-free controls. In contrast, sustained noxious forearm ischaemia produced a marked reduction in the intensity, unpleasantness and spatial distribution of pulpal pain (Sigurdsson & Maixner, 1994). Toothache does, however, affect gender differences in thermal pain detection and tolerance – such differences, evident in controls, were abolished in pulpitic pain patients, in as much as patients presenting with painful pulpitis showed no gender differences in thermal pain detection and tolerance thresholds (Edwards et al, 1999).

Toothache also affects behaviour. In a questionnaire-based investigation of 730 patients attending a dental hospital, in pain, over 80% reported a change in their normal behavioural pattern, including irritability, inability to concentrate, and sleep disturbance (Connor & Jeffrey, 1984).

In the only study of analgesia in toothache to be found, approximately one half of 114 children and adolescents, who received analgesics (typically paracetamol) for dental pain, experienced significant pain relief (Mason et al, 1997).

3.II.ii. **Post-Operative Dental Pain**

Unlike toothache, post-operative pain following third molar (wisdom tooth) removal is a commonly used model for analgesia. In a large meta-analysis of randomised double-blind trials on post-extraction pain, 10% of patients derived pain relief from the placebo, compared with, for example, 30% from oral tramadol (Moore & McQuay, 1997). Interestingly, pain relief in the dental patients was less than in other post-surgical patients (abdominal, orthopaedic, gynaecological), for all analgesics tested and placebo.

3.III. **AIMS**

A systematic review of the literature was conducted to provide answers to the following questions:

1. What psychological factors are associated with the report of dental pain?
2. Can psychological interventions affect the report of dental pain?

The search yielded forty-five studies.

3.IV. **REVIEW**

3.IV.i. **Categories**

The studies may be categorised into: a) clinical or experimental pain; b) with or without psychological manipulation.

Clinical pain models may be further divided into: i) toothache; ii) pain of dental treatment; iii) post-treatment pain.

Toothache studies involved self-report questionnaires and interview of patients attending general dental practice (GDP) or dental hospital (DH), some of whom were in pain.

Pain of dental treatment studies involved mostly adult patients attending GDP or DH, with pre- and post-treatment assessment principally of pain and anxiety.

Post-treatment pain most commonly followed third molar removal, and was assessed in conjunction with anxiety and other variables such as personality factors and psychological disorder.

Experimental tooth pain is produced by electrical stimulation, usually of upper incisor teeth. Detection and tolerance thresholds may be recorded. Evidently this procedure is standardised and may not produce the same feelings of anxiety as the clinical situation. The subject is likely to feel greater control over the proceedings.

Psychological manipulations: in general terms, the manipulations employed were intended to produce analgesia by increasing perceived control and decreasing anxiety.

3.IV.ii. **Subjects**

Apart from a few student studies, subjects were either consecutive dental patients or respondents to advertisements referring specifically to dental anxiety. In addition, two other patient groups were subjected to electrical tooth stimulation. American studies generally involved some form of financial incentive. Seven studies concerned children. Gender distributions varied, this being significant since gender differences in pain report were recorded.

3.IV.iii. **Design**

Correlational, cross-sectional and prospective designs were included, the latter generally being short-term, for example pre- to post-treatment changes.

One major issue in comparing pre-treatment pain expectancy with post-treatment report of pain experience is the consequent change in mood. The anxious patient is anxious pre-treatment at which time he/she will report high pain expectancy because that is the source of his/her anxiety. Similarly, anxiety is minimal immediately post-treatment in the absence of the threat of pain; pain experience was minimal because of the use of local anaesthetic and is therefore reported as such. The disparity between expectation and experience of pain during treatment is thus a direct function of anxiety.

Randomisation, blinding of investigators and dental staff to group, and inclusion of a control group were not universal.

3.IV.iv. **Pain Measures**

The use of electrical stimulation effectively means that subjective thresholds for pain detection and pain tolerance are elicited in response to an objectively measurable stimulus. Apart from a possible practice effect and the effect of past experience, these thresholds should be individually reproducible.

This is in stark contrast to clinical pain, where the stimulus is not standardised. It is impossible to objectively compare the cause of one person's toothache to that of another's, for example in terms of cavity size.

In modern dental treatment, the use of local anaesthetic means that, in the vast majority of cases, procedures are painless. Yet the dentist relies on the patient's

response, or lack of it, to what is supposedly noxious stimulation, in order to establish effective anaesthesia. Similarly, in the post-operative situation, one can record analgesic consumption but physiological variation in metabolism and personality differences in expression and communication mean that this is far from an objective measure.

The most widely used measures of pain are the visual analogue scale (VAS) and the Likert-type scale, both of which give a numerical rating. The VAS may be superior as a ratio scale. One criticism of such scales is that they are unidimensional despite the accepted multidimensionality of the pain experience. For this reason, separate scales may be used for 'intensity' and 'unpleasantness' of pain (see Houle,1988), but the validity of this distinction is unknown.

3.IV.v. **Psychological Measures**

Self-report questionnaires are favoured for their ease of use and their standardization, though validity and reliability are not always established, and the variety of questionnaires used makes comparison problematic. Child studies tend to involve observer ratings rather than self-report.

In the dental setting, the Dental Anxiety Scale (DAS, Corah, 1969), Dental Fear Survey (DFS, Kleinknecht et al, 1973), and Dental Beliefs Survey (DBS, Getz, in Milgrom et al, 1985), are valid and reliable measures of situation-specific anxiety (Corah et al, 1978; Johansson & Berggren, 1992; Kvale et al, 1997).

3.V. DISCUSSION OF FINDINGS

3.V.i. Studies on Clinical Pain without Psychological Manipulation (Table 3.I.)

Of the four studies on patients with toothache none had significant psychological findings. Weisenberg et al (1975) and Antczak-Bouckoms and Bouckoms (1985) used fairly crude measures of pain. Connor and Jeffrey's (1984) description of behavioural change in those in pain is from subjective retrospective report and is therefore unreliable. Kunzelmann and Dunninger (1990) found patients attending in pain to be more anxious and having more negative beliefs than those not in pain, which is unsurprising since it is those who are anxious who are more likely to put off a visit to the dentist until they are in pain.

Of the six studies on the pain of dental treatment, a consistent finding was that anxious patients had exaggerated expectation of pain whilst less anxious patients were more accurate in their predictions (Kleinknecht and Bernstein, 1978; Arntz et al,1990; Humphris et al,1991). Experience of pain, however, was not consistently related to anxiety. Kleinknecht and Bernstein's (1978) high fear group reported more pain than did the low fear group and Humphris et al's (1991) 'refuser' group apparently exhibited more pain, but Arntz et al (1990) found no association between anxiety and pain experience. The Kleinknecht study had a very poor initial volunteer response and the selection procedure is likely to have excluded the most anxious patients, ie. those given anti-anxiety medication. In the Humphris study the direct pain assessment measure of hand grip strength was based on anecdote alone.

Arntz et al (1990) described the need for several disconfirmatory experiences in order for anxious patients to become more accurate in their predictions of pain during treatment, which Kent (1986) felt was related to how typical the experience

was considered to be. Kent's study found no direct associations of change in anxiety with expected/experienced pain discrepancy nor with confidence in the typicality of the appointment.

The lack of effect of coping strategies on pain in Chaves and Brown's study (1987) was suggested to be due to the low pain levels involved in the treatment procedures (mean rating less than 2 on a 10-point scale). The study by Hargreaves et al (1983) was a small psycho-immunological investigation of individual variation in stress-provoked pain modulation.

Seven studies focused on post-treatment pain. Of these, four examined the predictive capacity of psychosocial factors on post-operative pain and recovery following third molar removal. Two studies found psychological factors, including pre-operative anxiety, to predict post-operative pain (George et al,1980; Feinmann et al,1987). Two further studies failed to show any such prediction (Hansson et al,1989; Gidron et al,1995). This discrepancy is likely to be due in part to the differing measures taken. Only Feinmann et al (1987) and Hansson et al (1989) used a standard validated psychological measure (GHQ) and only the former study specifically measured anxiety.

In the Feinmann study a regression analysis, in addition to correlation, would have informed regarding the relative predictive powers of the psychological variables. Hansson's study had the advantage of a standardised surgical procedure but, in recording on an hourly basis, may have incorporated too many measures. In Baume's study the post-surgical correlation of attention coping with pain does not imply prediction and the effect of coping on pain reduction is unclear. Gidron's study failed to control for analgesic consumption.

Table 3.I. Studies on clinical pain without psychological manipulation

a. Toothache

author (first)	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**findings**
Weisenberg	1975	emerg-ency clinic adult attenders in pain; ethnic differ-ences	75 (60% f) [1%]	direct questions re. current and expected pain	STAI (anxiety), PSI (anxiety physiologic) DAS (dental anxiety) interview and dentist ratings	no reported differences in current or expected pain between ethnic groups
Connor	1984	child and adult patients attending DH	1042 (43% f) ["few" refusers +1.5%]	numerical rating assess-ment	direct question re. behavioural attitude	82% of subjects experiencing pain felt their normal behavioural pattern to have been altered
Antczak-Bouckoms	1985	consecut-ive patients (aged 10 to 69 yrs) attending DH	61 (67% f) [10%]	simple report of pain and duration	IBQ	no significant difference on any scale of IBQ between 'acute pain' and 'no pain' groups; 'chronic pain' group differed

author (first)	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**findings**
Kunzel-mann	1990	random sample of GDP and DH adult patients	474 (56% f) [0%]	self-report	DAS, DBS (dental beliefs)	patients attending in pain more anxious [U=12644, p<0.001], and having more negative beliefs about the dentist [U=12569, p<0.001], than those not in pain
Edwards	1999	adult patients attending DH with pulpitic pain, and volunteer controls	46 + 33 (67, 61% f) [0%]	verbal descriptors and numerical rating	CSQ (coping strategies), STAI, POMS-Bi	pain patients showed higher anxiety (p<0.05), higher negative affect and lower positive affect, and higher catsatrophising. The group differences in affect remained one week after pain-relieving treatment.

b. Pain of dental treatment

author	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**findings**
Kleinknecht	1978	mostly adult patients attending GDP	128 (44% f) [89% of 4632 pt's contacted failed to reply, a further 8% not assessed, 22 given anti-anxiety medication and excluded	Likert-type scales for expected and experienc-ed pain	DFS (dental anxiety), AD (anxiety), WROS (behaviour) pre-treatment; PSI, DORS (behaviour) intra-treatment; AD several days post-treatment	no fear, sex or interaction effects on the differential between expected and experienced pain. Experienced pain [M=2.84] typically less than expected [M=3.19, $t(127)=2.39$, $p<0.05$]; high fear group reported more pain than low fear group [$F(1,80)=13.01$, $p<0.05$]
Hargreaves	1983	adult patients attending DH for third molar removal	9 (100% f) [0%]	VASs pre- and intra-op	VASs for anxiety pre- and intra-op	individual intra-operative pain, but not anxiety, correlated inversely with pre- to intra-operative rise in plasma endorphin-like immunoreactivity [$R=-0.72$, $p<0.05$]

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	findings
Kent	1986	adult patients attending GDP	125 (63% f) [25%]	VAS pre- and post-treatment and at 3 months	DAS pre-treatment and at 3 months	patients reporting a high discrepancy between expected and experienced pain, but not anxiety, less confident about the typicality of the appointment [$F(1,76)=8.40$, $p<0.005$]
Chaves	1987	adult patients attending DH and GDP	75 (57% f) [10%]	Likert-type scale post-treatment	STAI and DAS pre-treatment; TAS (hypnotic absorption), RLOC (locus of control) and Likert-type stress scales post-treatment	no difference in pain ratings between patients using coping strategies and those who denied cognitive activity, nor between deniers and catastrophisers
Arntz	1990	volunteer adult patients in GDP	40 (50% f) [23%]	VAS pre- and post-treatments (2) and at 5 months	DAS, VASs on aversiveness and anxiety	anxious dental patients tend to expect more pain than fearless patients and require several disconfirmations to become more accurate in their predictions; dental anxiety not associated with pain experienced

author	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**findings**
Humphris	1991	children (aged 7 to 16 yrs.) attending DH including uncoop-erative referrals from GDP's	58 (43% f) [9% excluded as uncoop-erative]	Likert-type scales for expected and experienc-ed pain; hand grip pressure	Likert-type scale for dental anxiety pre-treatment; videotape observation intra-treatment	overall expected pain [M=2.53] greater than experienced [M=2.12, $t(57)=2.17$, $p<0.05$]; 'refuser' group expected more pain than comparison group [$F(2,55)=4.07$, $p<0.05$]

c. Post-treatment pain

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	findings
George	1980	adults attending DH for third molar removal	38 (53% f) [3% refusal]	Likert-type scales and analgesic use for 4 days post-op; and duration	Likert-type scales for expectat-ions, anxiety, coping, HLC (health locus of control) pre-op.	higher levels of overall pain predicted by higher levels of anxiety about recovery [R=0.42, p<0.01], and more vigilant coping behaviours [R=0.28, p<0.05], even after controlling for surgical trauma [R=0.50, p<0.001]
Feinmann	1987	adult oral surgery patients attending DH	103 (61% f) [? refusers + 6%]	VAS at 1 and 3 days post-op. and analgesic use	STAI, VAS for anxiety, GHQ (psychological disorder) pre-op; EPQ (pers-onality), STAI post-op.	high trait anxiety [R=0.3, p<0.01], neuroticism [R=0.3, p<0.001] and psychiatric morbidity [R=0.3, p<0.01] predict persistent pain post-operatively
Hansson	1989	consecut-ive adult patients attending DH for third molar removal	100 (57% f) [0%]	VASs hourly post-op. for 72 hrs. and analgesic use	VASs for tension and stress; GHQ, BHS (hopeless-ness) pre-op; VASs again immediately post-op.	personality characteristics unrelated to post-operative pain

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	findings
Jones	1992	ortho-dontic patients at DH (aged 9 to 16 years)	43 (?% f) [4%]	VAS, yes/no questionn-aire, analgesic consumpt-ion (all post-treatment)	age differences only	older children reported greater pain [R=0.34, p<?]
Faucett	1994	adult patients attending DH for third molar removal	543 (55% f) [0%]	VAS post-extraction	ethnic differences only	women [t=-4.21, p<0.001] and younger patients [R=0.12, p<0.01] reported more pain; subjects of European descent reported less pain than those of black American [p<0.01] and Latino [p<0.05] descent
Baume (also published in Croog et al,1994 and 1995)	1995	volunteer perio-dontal patients attending GDP and DH (honorar-ium paid)	42 (100% female) [0%]	VAS, verbal rating scale, PAI (pain areas), duration and analgesic use post-treatments (2)	expected coping strategies, DAS, STAI, RGWAS (well-being), POMS (mood), MHLC (all pre-treatment); actual coping strategies, LAIS (impact) (post)	after allowing for complexity of surgery, attention coping (but not avoidance coping) correlated with pain [R=0.48, p<0.01]

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	findings
Gidron	1995	adolesc-ent patients attending DH for third molar removal	67 (55% f) [20%]	twice-daily Likert-type scale for 6 days post-op.	PANAS (negative affect), question re. expectancy of functional recovery, IBES (illness behaviour) for S's and parents, CISS (coping) (all pre-op.)	psychosocial factors did not predict pain

NB. See Key to Abbreviations (p. 261)

3.V.ii. **Studies with Psychological Manipulation (Table 3.III.)**

These studies were generally well-designed with adequate blinding of experimenters and dental staff and appropriate control groups. A wide variety of interventions were successful in producing some degree of analgesia in both children and adults, mostly undergoing routine dental treatment. Successful manipulations included audio-analgesia, emotive imagery, coping skills and sensory information, perceived control, distraction, progressive muscle relaxation, hypnosis, behavioural therapy, cognitive-behavioural therapy, and stress inoculation training.

Essentially the above techniques are designed to reduce anxiety and/or increase perceived control. These effects are achieved by relaxation and distraction or by attention to sensations. The relative merits of distraction and attention are unknown. Wardle (1983) found attention to sensations to be more effective in reducing pain and anxiety than was a visually interesting stimulus, but suggested that the latter was perhaps not adequately distracting. Wardle also used an unvalidated measure of anxiety. Siegel's studies found no difference in efficacy between conceptually distinct interventions: sensory information and coping skills (including relaxation). Individual differences in coping strategies may be influential, as in Sullivan and Neish's study (1999): disclosure of anticipated distress by writing down thoughts and feelings effectively reduced pain experience, during dental hygiene treatment, in catastophising patients.

Hypnosis would appear to have no additional benefit over simpler relaxation techniques (Katcher et al, 1984; Houle et al, 1988). The Houle study also lacked a control group. In the Morosko study, the lack of control group prevents assessment of any practice effect through repeated procedures. The reported increase in pain

threshold is likely to have been a function of anxiety reduction, in response to the suggestion of analgesia (implicit or explicit), and of perceived control.

The Enqvist study assessed pain solely by means of analgesic consumption, a crude measure and an active coping strategy in itself. In the Getka study, all interventions reduced anxiety and increased self-efficacy, and produced analgesia.

Table 3.II. Studies with psychological manipulation

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	manipulation	findings
Moros-ko	1966	dental students randomly selected from a group of volunteers	40 (all male) [0%]	tooth pain detection and tolerance thresholds in response to electrical stimulation, successive measurements	SHSS (hypnotic susceptibility)	audio-analgesia: music and 'white noise' with (1)explicit and (2)implicit suggestions re. alteration in feelings of pain	pain detection and tolerance thresholds raised with audio-analgesia [$p<0.05$] and especially with volume of white noise under the subjects' control [$p<0.01$]; no effect of implicit v. explicit suggestion nor of suceptibility to suggestion
Siegel	1980	children (aged 42 to 71 mnths) attend-ing pre-school prog-ramme for low-income families	42 (?% f) [0%]	?self-report; observer rating of response to local anaesth-etic injection	SPIES (internal-external) pre-session; VPT (anxiety), pulse-rate pre- and post-treatment BPRS (behav-iour) intra - treatment	3 conditions: (1)coping skills (general body relaxation, deep and regular breathing, pairing of relaxing cue words, imagery); (2)sensory information (re. procedure and typical sensations to be expected, drill sound); (3)control (story read)	children in coping skills and sensory information conditions displayed fewer disruptive responses [$p<0.01$], and rated as less distressed [$p<0.01$], than those in the control group

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	manipulation	findings
Siegel	1981	as above	26 (?% f) [0%]	?self-report; observer rating of response to local anaesth-etic injection	VPT, pulse-rate pre- and post-treatment; BPRS intra-treatment	as above	gains described above maintained during second treatment session c. one week later
Wardle	1983	adult patients attend-ing General Dental Pract-ice (GDP)	73 (53% f) [0%]	Likert-type scales post-treatment (dentist and patient ratings)	anxiety: 5-point scales post-treatment (dentist and patient ratings)	4 conditions: (1)sensation information (with specific reference to pain); (2)distraction (visually interesting stimulus); (3)perceived control (arm-raising encouraged as pause signal); (4)normal practice	lower pain and anxiety ratings in sensation information group cf. normal [$p<0.05$]; lower pain ratings in perceived control group [$p<0.05$]; no group differences in dentist's ratings

author	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**manipulation**	**findings**
Katch-er	1984	adult patients attend-ing DH for tooth extract-ion	42 (?% f) [?]	Patient Comfort Index	DAS, observer ratings, SHSS, BP and pulse rate	5 conditions: (1)aquarium contemplation (with tests of suggestibility - SHSS); (2)poster contemplation (as above); (3)poster contemplation with hypnosis (SHSS protocol); (4)aquarium contemplation with hypnosis; (5)non-intervention control (told to relax)	conditions (1), (3), (4) produced greater patient comfort than (2), (5) [$p<0.001$ to $p<0.06$]. Hypnosis did not augment the relaxing effect of aquarium contemplation

author	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**manipulation**	**findings**
Houle	1988	student volunt-eers	28 (50% f) [0%]	VASs for strength and unpleas-antness of pain	TAQ (hypnotic absorpt-ion)	2 conditions: (1)progressive muscle relaxation with suggestions for analgesia; (2)hypnotic induction with suggestions for analgesia	both conditions reduced the reported strength [$F(1,26)=15.46$, $p<0.001$] and unpleasant-ness [$F(1,26)=9.93 p<0.01$] of tooth pulp stimulation as well as pain detection threshold [$F(1,26)=6.64 p<0.01$], but not tolerance
Ander-son	1991	adult dental patients respon-ding to advert-isement	38 (58% f) [17%]	VASs for expected and experien-ced pain	STAI, VASs for anxiety and control pre- and post-treatment; APQ, Operator Rating Form post-treatment	3 conditions: (1)distraction (music) / suggestion of control; (2)distraction only; (3)no distraction	patients in both distraction conditions experienced less pain [$t(34)=3.59$, $p<0.01$] and more control [$t(34)=2.92$, $p<0.05$]

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	manipulation	findings
Getka	1992	dentally anxious adults (DAS>13) responding to advertisement	38 (c.50% f) [0% after exclus-ions]	Likert-type scales for expected and experien-ced pain	DAS, DFS-20 (pre-treatment and at 12 months); DSES (dental self-efficacy) pre-treatment Fear Thermometer, PSI, DORS intra-treatment	4 conditions: (1)behavioural (BT, 6 sessions - relaxation instruction, modeling, in vivo practice); (2)cognitive-behavioral (CBT, 6 sessions - Stress Inoculation Training, Quieting Reflex Training, attention-control techniques, in vivo practice of coping skills); (3)positive dental experience (PDE, benefits of, introduction to, and mangement by a dentist, identified by peers as being particularly effective in treatment of anxious patients; (4)waiting-list control (no intervention)	expected pain less after BT and CBT than after PDE and control [$F(3,32)=13.5$ $p<0.0001$]; experienced pain less after all interventions than after none [$F(3,32)=4.12p<0.01$]

author	**date**	**subject groups**	**numbers** (gender) [drop-out]	**pain measures**	**psychological measures**	**manipulation**	**findings**
Baron	1993	adult fee-paying endo-dontic patients attend-ing DH	188 (60% f) [c.5%]	VASs for expected and experien-ced pain ie. pre- and post-treatment	VASs for expected and experien-ced distress, STAI, IDCI (dental control)	2 conditions: (1)emotional-focus and (2)sensory-focus	relatively high pain experienced, by group high in desired 'dental control' but low in felt 'control', eliminated by sensory-focus manipulation but not emotional-focus [$F(1,176)=3.9 p<0.05$]
Heit-kemper	1993	children (8 to 11 years) attend-ing GDP	45 (?% f) [?]	0-10 rating of expected and experien-ced discom-fort	STAIC, (pulse rate), 0-10 belief rating of treatment (post-)	3 conditions: (1)paced respiration (with suggestion for relaxation); (2)cognitive coping (with suggestion for analgesia); (3) placebo story (with suggestion for analgesia and relaxation)	paced respiration most effective in reducing expected discomfort, cognitive coping more effective than placebo [$F(2,41)=20.4 p<0.001$]; state anxiety also reduced; experienced discomfort unaffected

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	manipulation	findings
Logan	1995	adult fee-paying endodontic patients attending DH	330 (61% f) [1%]	DDS pre- and post-treatment; 0-4 rating of experienced pain at one week	IDCI	4 conditions (pre-dental treatment): (1)sensory focus (physical sensations); (2)procedural information; (3) combination of (1) and (2); (4)no intervention	sensory focus reduced pain experience for the high desire / low felt control subgroup; procedural information did not add to this
Law	1994	adult volunteer fee-paying perio-dontal patients attend-ing DH	110 (55% f) [5%]	VASs for expected and experienced pain	STAI, IDCI pre-treatmentVAS for expected and experienced distress	2 conditions: (1)Stress Inoculation Training; (2)filler video (neutral)	expected pain unaffected; high desire/low felt 'control' patients in SIT condition experienced less pain than those in neutral condition [$F(1,93)=4.09 p<0.05$]

author	date	subject groups	numbers (gender) [drop-out]	pain measures	psychological measures	manipulation	findings
Enqvist	1997	adult patients on DH waiting list for third molar remov-al	69 (52% f) [4%]	post-op VAS and analgesic use	VASs for anxiety 3 weeks and 30 mins. pre-op; VAS for post-op well-being	2 conditions: (1) hypnotic relaxation induction audiotape with suggestion for healing and analgesia, practice; (2)no intervention	preoperative level of anxiety maintained on day of surgery after intervention, whereas anxiety increased in the control group [p=0.002]; consumption of 3 or more analgesic doses (daily average) less after intervention [3% of patients] than in control group [28%, p=0.005]
Sullivan	1999	students; dental hygiene treatment	80 (65% f) [0%]	VAS post-op	PCS, DAS-R, POMS pre- and post-op	2 conditions: (1)disclosure of expected distress, thoughts and feelings; (2)control - description of previous day's activities	catastrophisers reported more pin than non- ($p<0.05$) in the control group only. Disclosure reduced catastrophisers' pain ($p<0.05$)

NB. See Key to Abbreviations (p. 261)

3.VI. MANAGEMENT OF DENTAL ANXIETY AND PAIN

There remains debate regarding the most effective strategies in managing dental anxiety, in terms of distraction or attention. Lindsay et al (1985) have argued that provision of information, regarding the sensations to be expected during and after treatment, is ineffective and inappropriate in dentistry, unlike other medical procedures (Suls & Wan, 1989).

Information regarding post-operative sequelae and pain management increases pain relief and satisfaction with pain control following third molar extraction, compared with control information on wound healing (Vallerland et al, 1994).

Gentry (1997) found the provision of a positive pain coping strategy to increase perceived efficacy of control over pain following third molar removal, whilst having no apparent effect on anxiety.

Suitable coping strategies for anxious dental patients may be encouraged through the use of hypnotic techniques (Shaw & Niven, 1996). Heitkemper et al (1993) were able to reduce anxiety and expected discomfort, but not reported pain, in children. They provided one of two brief treatments by audiotape: paced respiration and cognitive coping instructions; both were effective over a placebo recording.

Moore et al (1996) compared hypnotherapy with group therapy and individual systematic desensitization in the management of 174 extremely dentally anxious subjects (DAS > 15), and found all three techniques to be efficaceous in reducing dental anxiety (DFS) and increasing trust of the dentist (DBS), despite high drop-out rates after one year (c.50%).

A long-term review (1-4 years) of 23 subjects, who had successfully completed a 4-session group behavioural programme, found 70% to be paying regular dental visits

(at least an annual check-up) (Liddell et al, 1994). The non-attenders had remained more anxious than the attenders on completion of the treatment programme, they were less satisfied with their dental visits, and they had experienced a higher proportion of invasive to non-invasive dental procedures subsequently. The authors discussed the heterogeneity of dentally anxious individuals and the multiplicity of factors likely to predict the maintenance of dental anxiety and avoidance.

3.VII. CONCLUSIONS

From this systematic review of the relationships between psychological factors and acute dental pain, several conclusions may be drawn:

1. Studies of the effect of psychological factors on toothache are inconclusive. The treatment of choice for the acute pain condition of pulpitis is surgical restoration. Yet many individuals continue to fear dental treatment, and this fear perpetuates avoidance of dental treatment, which would resolve the pain. Investigation of psychological mediators of toothache might provide targets for manipulation, analgesia and anxiety management, helping to reduce dental avoidance and to improve dental health.

2. Studies on the pain of dental treatment consistently find pain experienced less than expected. This highlights the continuing need for health education to emphasise the painlessness of modem treatment, and for dental undergraduate training to encourage painless techniques.

3. Pain and anxiety are inextricably linked, since the report of both expectation and experience of pain is influenced by current mood, including state anxiety..

4. The influence of coping strategies upon dental pain is uncertain. Coping research has tended to concentrate on chronic pain conditions. There is a need for research to identify adaptive and maladaptive coping strategies in acute pain as change, through instruction, may be relatively simple and cost-effective.

5. Studies on the effect of psychological factors on post-surgical pain are equivocal. This is likely to be due to methodological differences and more work is needed on this pain model.

6. Studies of experimental pain indicate pain detection to be influenced by anxiety, whilst pain tolerance is less influenced. Pain detection threshold is perhaps more relevant to the clinical situation than tolerance threshold since clinical pain levels are minimal, although the generalisability of experimental pain paradigms, to the clinical setting, is questionable.

7. Interventions that measurably increase feelings of control and relaxation help to reduce the reported pain of dental treatment. Pre-treatment disclosure of anticipated distress may also be of benefit to individuals with a tendency to catastrophise.

8. Given the success of psychological manipulations in moderating acute pain within the dental setting, a logical extrapolation is to develop an intervention for chronic facial pain, based on anxiety reduction and augmentation of perceived control.

Chapter 4. CHRONIC HEADACHE

As with TMD, the causes of tension-type headache and migraine remain unknown, yet distress and disability have been investigated in headache. Unlike other chronic pain conditions such as low back pain, in which mobility is directly impaired, headache is more indirectly disabling and is therefore an appropriate condition with which to compare TMD on these parameters.

4.I. EPIDEMIOLOGY

The lifetime prevalence of headache is probably close to 100% (Ho et al, 1997) and nearly three quarters of the population report recent head pain (for example: Koutantji et al, 1998). Using International Headache Society (IHS) criteria, the prevalence of headache over 12 months in the general adult population is approximately 10% for migraine and 20 to 30% for frequent (more than monthly) tension-type headache (Rasmussen & Olesen, 1994), yet only one half of migraine sufferers, and less than one sixth of tension-type headache sufferers, seek medical advice (Rasmussen et al, 1992).

Frequency and severity of symptoms may be the chief factors in determining treatment-seeking but these variables are evidently subjective and, hence, psychological factors may well be influential. Clinic patients report greater occupational disability than do headache sufferers not seeking treatment, even after controlling for headache severity (Ziegler & Paolo, 1996).

There is a female preponderance in both migraine and tension-type headache: approximately 1:2.5 (male-to-female ratio) and 1:1.5 respectively, in the general population (Rasmussen & Olesen, 1994). These sex differences may be due to hormonal or psychological influences but, in any event, are compounded by the greater use of health services by women. Ethnic and cultural differences also affect pain report, for example Americans of European descent report less postoperative dental pain than do those of Black American or Latino descent (Faucett et al, 1994). Migraine has, in the past, been considered an illness of the professional classes, and this may reflect a barrier to consultation in low-income groups (Lipton et al, 1992).

4.II. DIAGNOSIS

Diagnosis of a particular class of headache is made on the basis of self-report of subjective symptoms and elicitation of signs through apparently objective testing. There is good evidence for a fundamental distinction between migraine and tension-type headache (Rasmussen & Olesen, 1994). The former appears to be the manifestation of a basic neurochemical disorder, whilst the latter has distinct relations with stress. The occurrence of tension-type headache in migraineurs should not, therefore, present any conceptual problem (Ulrich et al, 1996). However, psychophysiological tests, including frontalis electromyography (EMG), temporal blood volume pulse (BVP), temporal and finger skin temperature, fail to differentiate between the two syndromes (Lichstein et al, 1991) and one is left, once again, with self-report.

4.III. SPECIALIST REFERRAL

Referral by a General Practitioner to a Neurologist is followed by a lengthy spell on a waiting list, necessitated by budgetary constraints and the relative lack of urgency. Specialist pain clinic populations exhibit greater prevalence of psychiatric morbidity than do population-based or primary care samples (von Korff & Simon, 1996). This suggests either: 1. it is only those individuals who find it difficult to adjust to their pain, and are consequently distressed, who end up in specialist clinics; or 2. individuals become distressed over the time it takes to get them there. The lack of prospective studies of chronic pain precludes any conclusions.

The problem with exhaustive testing such as EMG, vascular studies and imaging, is the reinforcement of the medical model of illness. With each negative test, the patient may well become convinced of the existence of an undetected organic lesion. It has recently been suggested that "greater and earlier exposure to the primary health-care setting would modify doctors' beliefs about the appropriateness of extensive physical investigation, and the relative values of clinical histories and physical examination findings" (Peveler, 1998, p.95). A neuroimaging examination is only indicated by an atypical clinical picture, which is rare (Jelencsik, 1998). Neurologists may view pain relief as the most important aim of management and fail to provide adequate explanation and reassurance (Edmeads, 1998). Yet reassurance by itself may actually prove harmful in attempting, via verbal and non-verbal cues, to persuade the patient of the absence of disease (Coia & Morley, 1998). In any event, a busy Neurology outpatient clinic does not lend itself to patient listening to what may be a lengthy and complex history, nor to advice and counselling thereon.

Ideally a multidisciplinary team should undertake the management of headache patients but this may not be financially practical. Certainly the Liaison Psychiatrist has much to contribute in the elicitation and interpretation of a pain history, and in the informed and informative provision of anti-depressant medication as an aid to management.

4.IV. PERSONALITY AND MIGRAINE

As with TMD, there is no evidence for a typical migraine personality (Kohler & Kosanic, 1992). Migraineurs do, however, exhibit greater neuroticism than controls (Leijdekkers & Passchier, 1990; Breslau et al, 1996; Persson, 1997). Yet the concept of the neurotic personality might be better considered as a tendency to perceive greater stress than others, and again, in the absence of prospective studies, one cannot determine whether this tendency derives from the experience of migraine or vice-versa.

Personality testing of a Chinese migraine sample found increased neuroticism-anxiety, on a translation of the Zuckerman-Kuhlman Personality Questionnaire (ZKPQ), relative to controls and post-traumatic headache (Wang et al, 1999). The authors speculated a shared biological background (low 5-HT activity) for anxiety and migraine.

There may well be a role for previous experience of pain and distress in predisposing adults to symptoms. A significant positive relationship has been shown between depression and a history of childhood sexual and physical abuse in patients with chronic pain (Goldberg, 1994). The familial occurrence of headaches is likely to be

encouraged by modeling, especially in the absence of any evidence for a genetic basis (Bahra & Goadsby, 1998).

4.V. A SHARED BIOLOGICAL ORIGIN WITH DEPRESSION

The risk of developing depression in migraineurs, relative to population controls, is virtually identical to the risk for developing migraine in depressives (3.2 and 3.1 respectively, Breslau et al, 1994). Together with evidence of comorbidity from familial and biochemical studies and the therapeutic efficacy of anti-depressant agents in migraine, this association has led to the postulation of a common pathophysiological mechanism for migraine and depression (Bahra & Goadsby, 1998). The risk of depressed individuals developing migraine is greater than their risk of developing other severe headache, strengthening the likelihood of a shared biological origin for the former (Breslau et al, 2000). The increased risk of major depression in headache sufferers strengthens the need for psychiatric liaison but does not support the psychodynamic notion of migraine as masked depression (Blumer & Heilbronn, 1982).

4.VI. THINKING AS A CHRONIC PATIENT

Tension-type headache sufferers show mildly more anxious and depressed moods (Hatch et al, 1991), much greater fear of severe pain (Hursey & Jacks, 1992), and more frequent suppression of anger (Hatch et al, 1991), than do controls.

Chronic Daily Headache, considered to be transformed migraine in many cases, is associated with high anxiety levels (Mongini et al, 1996); whilst migraine is associated

with depression (Verri et al, 1998). Rates of suicidal ideation and suicide attempts are increased in migraine with aura, especially with coexisting depression (Breslau, 1992). Quality of life studies have demonstrated the impact of recurrent headaches. The burden of migraine may be equal to, or worse than, that of arthritis, diabetes or low back pain (Solomon, 1997). It would appear to be the emotional component of the pain which predicts a fall in health-related quality of life, rather than headache diagnosis, frequency or severity (Passchier et al, 1996).

Although there is no evidence of associated cognitive impairment *per se* (Leijdekkers et al, 1990), Demjen and co-workers have described a cognitive shift with headache of increasing severity "whereby the patient's primary concern moves from situational and interpersonal distress to distress associated with the disorder itself" (Demjen et al, 1990, p.427). They stress the impact of both symptom intensity and duration in increasing headache-related distressing thoughts and feelings.

Other chronic conditions show a relationship between illness perceptions, disability and mood. Chronic fatigue syndrome patients, who consider their conditon to be serious and beyond control or cure, report greater physical, social and mental health impairment (Heijmans, 1998). Similar relationships may exist in chronic headache patients but have not been investigated.

4.VI.i. **Stress and Coping**

The perception of stress is an important factor in symptom recurrence, certainly in tension-type headache (De Benedittis & Lorenzetti, 1992). Both life-events and daily 'hassles' have been studied, and it would appear that, whilst life-events may trigger hassles, it is the perceived severity of those hassles that best predicts headache

frequency and intensity (Fernandez & Sheffield, 1996). Physiological disregulation of stress response may be involved in tension-type headache but not in migraine (Davis et al, 1998), although migraineurs show increased cardiovascular activity in response to stress compared with controls (Stronks et al, 1998). Tension-type headache sufferers may fail to pay adequate attention to environmental information when appraising stressful events; whilst migraine may be associated with delayed recovery of cardiovascular response to stress (Holm et al, 1997). It may be the ways in which headache sufferers respond to stressful events that determine the onset and intensity of attacks (Marlowe, 1998); and the relationship between life stress and headache frequency may be stronger in women (Reynolds & Hovanitz, 2000).

There is likely to be a complex temporal relationship between stress, mood and migraine (Spierings et al, 1997). In coping tests, migraine patients appear to be more negative as to their anticipated future activities than do cluster headache patients (Blomkvist et al, 1997). In female migraineurs, headache is associated with stress during the premenstrual and ovulatory phases. This supports a relationship between the menstrual cycle, the stress-appraisal-coping process, and migraine (Holm et al, 1996).

4.VII. TREATMENT

Successful treatment of headaches is associated with reduction in disability, improvement in quality of life, amelioration of mood, positive adaptation of coping strategies in response to stress, and enhanced perceived control over pain. These are not all to be achieved with a simple pill. For a drug to be effective in reducing the pain of headaches, its prescription must be accompanied by expectation of its

efficacy (Voudouris et al, 1990). Consequently, should that efficacy fail to meet expectation, the pain may become less responsive at the next attempt.

The doctor-patient interaction plays an important role in nurturing expectation but subtle cognitive and conditioned responses are also likely to be involved (Wall, 1994). Placebo response is greatly enhanced by the experience of effective analgesia (Voudouris et al, 1990) and, hence, an individual's belief in the efficacy of any therapy is greatly enhanced if it appears to improve any aspect of the pain, even if that improvement cannot ultimately be attributed to the therapy itself but may in fact be a spontaneous remission. This may account for some of the effectiveness of anti-depressant agents in chronically recurrent pain, including headache. Even in an apparently non-depressed pain patient, such agents can be expected to affect mood, which is an aspect of the pain experience, thus breaking the cycle of stress, pain, disability and distress. Pain is adversive and depression may be the result of adversity, so the finding of a shared neurochemistry involving serotonin is not altogether surprising.

Tricyclic anti-depressants, notably amitriptyline, have been established to be of benefit, at least in the short-term, in reducing the duration of daily tension-type headaches, whilst having less effect on more episodic complaints (Gobel et al, 1994; Cerbo et al, 1998). However, when strict criteria for improvement in duration, frequency and intensity are employed, the effect of amitriptyline is no greater than placebo (Pfaffenrath et al, 1993). In addition, despite the difficulties in comparing such disparate treatment modalities, cognitive behavioural therapy has been shown to be slightly more effective than amitriptyline (Holroyd et al, 1991). Interestingly, withdrawal of amitriptyline appears to result in elevation of frontal EMG levels in

tension-type headache sufferers compared to healthy controls, whilst no difference was evident during medication (Ellertsen et al, 1987).

Regardless of treatment modality, psychological factors are contributory. Treatment effects have been shown to be modestly related to changes in coping strategies and appraisal processes (ter Kuile et al, 1995), and response, for example to sumatriptan therapy, is partly determined by pretreatment quality of life (Litaker et al, 1997), whilst anxiety predicts endurance of symptoms in both migraine and tension-type headache (Guidetti et al, 1998).

4.VII.i. **Cognitive Behavioural Therapy**

Both cognitive therapy and behavioural self-management training are effective in decreasing headaches and depressive symptoms (Martin et al, 1989). The improvements in symptoms and mood cannot be separated. A meta-analysis has revealed substantial support for the equivalent effectiveness of both propranolol and biofeedback / relaxation training (Holroyd & Penzien, 1990). Biofeedback techniques may be no more effective in the short-term than relaxation training but both are more effective than control conditions, although this may be a function of therapist contact (Chapman, 1986). Biofeedback may augment long-term improvement after autogenic relaxation training (Cott et al, 1992), and, by demonstrating the influence of thoughts and emotions on bodily reactions, may prepare the way for successful cognitive treatment (Kropp et al, 1997). A recent study has suggested that a mass media self-help programme for the behavioural treatment of chronic headache may be effective, across diagnostic groups, in reducing frequency and medication use (de Bruijn-Kofman et al, 1997).

There is a fundamental difficulty with pain report as both diagnostic criterion and outcome determinant. Pain is subjective and influenced by mood changes, however transient. Instruments such as the migraine-specific quality-of-life measure (MSQOL: Wagner et al, 1996), encompassing psychosocial as well as physical functioning, may provide more objective markers of illness and treatment response.

The next chapter will review the assessment of coping strategies and their association with pain, disability and distress in various pain groups.

Chapter 5. COPING IN ACUTE AND CHRONIC PAIN

5.I. INTRODUCTION

Coping has been defined as 'purposeful effort to manage or vitiate the negative impact of stress' (Jensen et al, 1991). The Cognitive Transactional Theory of Stress (Lazarus, 1975) considers coping as the third stage in the reaction to stress, the first being primary appraisal, or perception of a threat, and the second being secondary appraisal, or conceptualising a potential response to the threat (Lazarus & Folkman, 1984). In the face of stress, individuals appraise the situation to assess its personal relevance, and then engage in secondary appraisal, whereby they assess whether they can act to reduce the harmfulness of the stressor.

Individuals' coping strategies are considered to play a role in the development of chronicity, the maintenance of pain symptoms, and the adjustment to chronic pain (Jensen et al, 1991). The relative degrees to which an individual employs adaptive or maladaptive strategies are thought to explain some of the variance in affect and physical disability seen in chronic pain populations. Investigation of coping strategies correlationally and longitudinally might therefore provide a prediction of pain patients' progress as well as an evaluation of treatment outcome, if associated with distress and disability.

5.II. LITERATURE REVIEW

Jensen et al (1991) published a comprehensive review of the research to that date into beliefs, coping efforts and adjustment to chronic pain, and emphasised the

preponderance of correlational studies which fail to establish causal relationships included in the theoretical models of pain as a stressor. Subsequent longitudinal studies and clinical trials of treatment modalities have demonstrated modest associations, of pre-treatment to follow-up changes in coping, with patient improvement (Turner et al, 1995; ter Kuile et al, 1995a; Nicassio et al, 1995).

5.III. INSTRUMENTS

5.III.i. **The Ways of Coping Checklist (WCCL)** (Folkman & Lazarus, 1980)

This scale was based on the authors' Cognitive Transactional Theory of Stress. The original scale had 68 items with 'yes' / 'no' responses. This was revised to the Ways of Coping Questionnaire by the authors in 1985, comprising 66 items with a Likert scale of 0 to 3 response, whereby patients endorsed the applicability of each item to their situation. A final revision included 42 items.

The scale has five subscales: problem-focused coping; self-blame; avoidance; wishful thinking; and seeking social support. Vitaliano et al (1985) found the scale to have good construct and concurrent validity but little convergent validity. Factor analyses have shown between two and nine clusters of coping strategies. Folkman and Lazarus (1980) described 91% agreement between raters, demonstrating reliability.

5.III.ii. **The Vanderbilt Pain Management Inventory (VPMI)** (Brown & Nicassio, 1987)

This is a twelve-item scale comprising two subscales of 'active' (adaptive) and 'passive' (maladaptive) coping strategies. Active strategies include exercise, activity and ignoring pain. Passive strategies include rest and medication use.

5.III.iii. **The Coping Strategies Questionnaire (CSQ)** (Rosenstiel & Keefe, 1983)

This 42-item scale involves seven subscales: diverting attention; reinterpreting pain sensations; use of coping self-statements; ignoring pain sensations; praying or hoping; catastrophising; increasing activity levels. Snow-Turek et al (1995) used the VPMI and CSQ to demonstrate the validity of active and passive coping dimensions, whilst finding the CSQ to be the more psychometrically sound scale.

Factor analyses have been inconsistent across studies and patient populations but Lawson et al (1990) suggested three basic factors: conscious cognitive coping; self-efficacy beliefs; pain avoidance. The catastrophising and behavioural coping subscales have not loaded consistently on any one of these basic dimensions. Swartzman et al (1994) found five distinct, consistent and valid factors, reintroducing 'catastrophising' as one of them. McCracken and Gross (1993) considered catastrophising to be a distress response.

5.III.iv. **The Coping Scale (CS)** (Holmes & Stevenson, 1990)

This scale measures two types of strategies: attentional (information-seeking, viewing circumstances more favourably, etc.) and avoidance (thinking of something pleasant, eating more, etc.).

5.III.v. **COPE** (Carver & Scheier, 1989)

The self-administered scale has two formats: dispositional and situational; with thirteen conceptually distinct subscales based on the theoretical literature. Five subscales (four items each) measure problem-focused coping: active coping,

planning, suppression of competing activities, restraint coping, seeking instrumental social support. Five subscales (four items each) measure emotion-focused coping: seeking emotional support, positive reinterpretation, acceptance, denial, turning to religion. Three subscales (three items each) measure: venting of emotions, behavioural disengagement, mental disengagement.

Validity of the scale was assessed by factor analysis of the results of a large group of students. Twelve factors were identified, all in accord with *a priori* assignment of items to subscales. In terms of reliability, correlation between scale items is generally less than 0.30 but the authors claim this as support for the empirically distinct domains of the scales.

Endler and Parker (1990) have criticised the scale for over-emphasising gender differences and have mentioned the psychometric problems with four-item scales, namely the lack of an undecided mid-point.

5.III.vi. **The Coping with Health, Injuries and Problems scale (CHIP)**

(Endler et al, 1992)

This instrument has been developed for use across diverse populations to provide measures of emotion-focused and task-oriented responses to injury, and was considered useful in chronic musculoskeletal pain patients, overlapping the CSQ but also providing information on palliative and instrumental coping responses, and being shorter (Hadjistavropoulos et al, 1999a). Its usefulness in idiopathic conditions is, however, questionable in the absence of identifiable injury.

5.III.vii. **The Chronic Pain Coping Inventory (CPCI)** (Jensen et al, 1995)

This scale has been validated in a group of patients referred to an interdisciplinary treatment programme, and found to have greater predictive ability than the CSQ in its relationship with pain severity (MPI) (Hadjistavropoulos et al, 1999b). The results did confirm the predictive properties of the Catastrophising subscale of the CSQ but this led the authors to question the value of the remaining CSQ subscales, and to rekindle the debate over whether catastrophising is a coping strategy or an appraisal.

5.III.viii. **Critique**

A number of criticisms have been raised against the instruments used to date.

Endler and Parker (1990) consider the WCCL to have been used more often by researchers than its psychometric properties justify. Carver et al (1989) felt WCCL items to be ambiguous. The WCCL involves a self-defined stressor. This gives rise to problems in the assessment of chronic pain patients. These individuals are 'coping with what?' (Fordyce, 1991). Pain may not be the most salient stressor when multiple stressors such as loss of income, confinement and marital discord arise. Keefe et al (1992) recommended specifying the stressor to the patient.

The VPMI and CSQ were specifically designed to assess coping strategies used to manage pain. With regards to the VPMI, Keefe et al (1992) have asked whether any coping strategy is truly 'passive', since active compliance and decision-making are required, for example, in medication use. This may highlight an over-simplification. The composite scoring of the VPMI allows for better statistical results but at the expense of specificity of individual strategies.

Certain items in the CSQ are, strictly speaking, appraisals rather than coping strategies; for example: the 'self-efficacy belief' and 'catastrophising' factors. Jensen et al (1991) mentioned the lack of cross-validation of the CSQ's internal structure, though this issue has been subsequently addressed by Swartzman et al (1993). Keefe et al (1992) also discussed how the CSQ was originally designed to measure the *frequency* with which patients utilised a variety of cognitive and behavioural skills, as well as their perceived *effectiveness* in controlling and reducing pain, whilst these values have generally been confounded.

5.IV. FINDINGS

Table 5.I. includes studies from 1990 until September 2000 of coping strategies in chronic pain. **Table 5.II.** includes the few studies on acute pain.

5.IV.i. Ways of Coping Checklist (WCCL)

Five studies have indicated that WCCL items are associated with measures of psychological and physical functioning in chronic pain patients, especially the association between 'wishful thinking' and poor functioning (Turner et al, 1987; Manne & Zautro, 1990; Felton et al, 1984; Parker et al, 1988; Regan et al, 1988).

Ferrer-Perez and Truyols Taberner (1996) used a Spanish version of the WCCL developed by Rodriguez-Marin et al (1992) in a small group of low back pain patients. They found the use of coping strategies to be associated with increased pain whilst, in relation to emotional factors, increased active strategies and reduced avoiding strategies were associated with less anxiety and depression.

5.IV.ii. **Vanderbilt Pain Management Inventory (VPMI)**

Using the VPMI, active coping has been found to be associated with less pain, depression and disability whilst passive coping is associated with higher levels of these factors (Brown & Nicassio, 1987). A further study found a moderating influence of pain level, in that low pain levels removed the relationship between passive coping and depression (Brown et al, 1989). A longitudinal predictive value for depression and disability over six months was demonstrated.

5.IV.iii. **Coping Strategies Questionnaire (CSQ)**

Two studies with the CSQ found a significant relationship between ignoring pain / coping self-statements and pain severity (Hagglund et al, 1989; Parker et al, 1989). Ten studies, however, found none (Beckham et al, 1991; Gil et al, 1989; Gross, 1986; Keefe et al, 1987a;b; 1990a;b;c; Rosenstiel & Keefe, 1983; Spinhoven et al, 1989). Six out of nine studies found a significant inverse relationship for ability to control and reduce pain load with pain intensity and functional disability.

Jensen and Karoly (1991) found a good correlation with adjustment while controlling for pain severity. Dozois et al (1995) found the predictive value of factor scores to be dependent on the measures of adjustment. As an individual score, catastrophising predicted psychological distress. Jensen et al (1993) found catastrophising to be dependent on pain site, educational level and gender. Hill et al (1995) found catastrophising to be associated with increased pain report in amputeees with phantom limb pain.

A study by Haythornthwaite et al (1998) has found associations between positive adaptive coping strategies and perceived control over pain. In a mixed group of

chronic pain patients, including unspecified facial pain, coping self-statements and reinterpreting pain sensations (CSQ) predicted greater perceptions of control over pain, as measured by the Survey of Pain Attitudes (SOPA), after controlling for pain severity and education, whereas ignoring pain sensations predicted lesser perceived pain control. Ignoring pain sensations was therefore construed as a maladaptive strategy. Catastrophising was excluded from the study because of the debate over whether it should be considered a coping strategy as such.

In a large study of low back pain patients, Main et al (1996) found different strategies to be more clearly identified at different stages in chronicity. Another study divided a tertiary care sample of chronic low back pain patients into subgroups on the basis of CSQ scores, deriving three clusters ('cognitive coping', 'low response', 'catastrophising / distraction') differing significantly across measures of pain (MPQ), distress (BDI) and physical functioning (MPI) (Riley et al, 1999).

5.IV.iv. **Coping Scale**

Holmes and Stevenson (1990) found those chronic pain patients using *attentional* strategies on their Coping Scale to be less depressed and anxious and more active than the others, whilst those acute pain patients using *avoidance* strategies were less depressed and anxious and more active than the others.

5.IV.v. **Other Groups**

In populations other than chronic pain groups, Lefebvre et al (1995) found the CSQ to be a reliable measure for the study of pain-coping strategies in a student population. Buckelew et al (1992) used an electrodiagnostic model, eliciting pain and

anxiety in a group of fifty patients. Using a post-procedural situation-specific version of the CSQ, they found catastrophising, diverting attention and self-statement to positively correlate with pain whilst reinterpreting was a negative correlate.

In an acute model of pain during dental hygiene treatment, catastrophisers were seen to benefit from disclosure, of anticipated thoughts and feelings, in terms of subsequent pain and distress (Sullivan & Neish, 1999). This prospective study used the Pain Catastrophizing Scale (Sullivan et al, 1995) in a group of students, and was considered as support for the clinical use of such disclosure in identified catastrophisers, despite the student sample being unrepresentative of a clinical population.

5.IV.vi. **Further Developments**

A novel redefinition of chronic pain is as *chronic interruption* of thought and behaviour; coping with chronic pain is envisaged as an "ongoing attempt to recover from chronic interruption by repeatedly switching between pain and other demands in the environment" (Eccleston & Crombez, 1999: p.363). This concept brings coping and disability closer together, as the effectiveness of the former determines the degree of the latter; disability being the interference of pain switching the individual away from his/her activities.

Work examining moderating factors on the effects of coping strategies, such as pain severity, gender, diagnosis and pain duration, is of particular interest (for example: Jensen et al, 1993; Main et al, 1996), throwing light on issues such as female predominance in chronic pain groups, and the acute-to-chronic transition.

Table 5.I. Studies that have examined the relationship between coping strategies and adjustment to chronic pain:

Author	Sample	Design	Measures Coping	Adjustment	Results
Schnurr et al (1991)	100 TMD patients	correlational	WCS®	symptoms	WCS significant predictor of pain intensity (51%)
Ferrer-Perez & Tabemer (1996)	25 low back pain patients	correlational	CEA (WCCL)	HAD pain report	use of approximate or avoiding strategies associated with more pain but increased active and reduced avoidance strategies associated with less anxiety and depression
Jaspers et al (1993)	53 TMD patients	correlational	CSSQ	MPI, GHQ symptoms	expression of emotions and wishful thinking associated with more pain, suffering, distress
Snow-Turek et al (1995)	76 chronic pain patients	correlational	CSQ, VPMI	MPI, CES-D HSCL-21	active and passive coping dimensions valid and clinically useful
Barkwell (1990)	100 cancer pain patients	correlational	CSQ	MPQ depression	strongest impact on pain, depression, and coping scores made by meaning ascribed to pain
Jensen et al (1993)	121 neck, shoulder, back pain patients	correlational	CSQ	pain report, GSI, disability, MPI	catastrophising of spinal pain in lower educational levels, females only
McCracken & Gross (1993)	165 low back pain patients (mostly)	correlational	CSQ	pain, anxiety symptom scale	cognitive anxiety reduces coping, physiological anxiety increases coping; catastrophising as a distress response?

Author	Sample	Design	Measures Coping	Adjustment	Results
Swartzman et al (1993)	126 whiplash patients	correlational (factor analysis)	CSQ		5 distinct, consistent and valid subscales
Dozois et al (1995)	200 low back pain patients	correlational	CSQ	Oswestry functional status, GSI	factor scores or individual scores better predictors depending on definition of adjustment. Catastrophising predicted psychological distress
Hill et al (1995)	amputees with phantom limb pain	correlational	CSQ	pain report	catastrophising associated with more pain
Lefebvre et al (1995)	252 students	correlational	CSQ	pain survey	CSQ reliable in young adult population; greater perceived efficacy v. pain groups
Main et al (1996)	517 low back pain patients	correlational	CSQ	chronicity	different strategies in different groups
Keefe et al (1997)	130 osteo-arthritis patients	correlational	CSQ	ASES, MPQ	ignoring pain sensations and coping self-statements related to higher self-efficacy; catastrophising related to lower self-efficacy
Haythorn-thwaite et al (1998)	195 chronic pain patients	correlational	CSQ	MPI, SOPA	adaptive (coping self-statements, reinterpreting sensations) and maladaptive (ignoring pain) strategies identified, relating to perceived control over pain.
Cathcart & Materazzo (1999)	64 headache sufferers, community	correlational	CSQ	STAI, MPI	artificial neural network model used to predict headache. Ignoring sensations, coping self-efficacy, and reinterpreting mot significant predictors.

Author	Sample	Design	Measures Coping	Adjustment	Results
Riley et al (1999)	975 pain clinic patients	correlational	CSQ-R	BDI, MPI, MPQ, pain VRI	3 clusters identified: cognitive copers, low raters of CSQ-R items in general, and catastrophisers / distracters
Keefe et al (2000a)	168 osteo-arthritis patients	correlational	CSQ	AIMS, SCL-90	women had higher levels of pain and disability than men; catastrophising mediated the relationship between gender and pain-related outcomes
Jensen et al (1994)	111 leg, back, head pain patients	correlational	CPCI	CES-D, MPI PDS	CPCI scales reliable. Guarding, resting, asking for assistance, task persistance most closely related to functioning
Turner et al (2000)	169 pain clinic patients	correlational	CSQ, CPCI	VAS, Roland Scale, CES-D	coping scores independently predicted physical disability but not depression; catastrophising independently predicted depression but not physical disability
Potter & Jones (1992)	45 acute musculo-skeletal pain pts. in Primary Care	longitudinal	interview, VPMI	pain VAS, MPQ, Goldberg's questionnaire for anxiety and depression	high pain intensity, depression and passive coping associated with development of chronic symptoms?
Sundblom et al (1994)	24 chronic pain patients	longitudinal	CSQ	BDI, IASP-database outline	no significant change in scores

Author	Sample	Design	Measures Coping	Adjustment	Results
Nicassio et al (1995)	69 fibro-myalgia patients	longitudinal	CSQ	pain, depression PMI pain behaviour	high active coping and low pain control / rational thinking predict more pain and depression
ter Kuile et al (1995)a	144 headache patients	longitudinal	CSQ	HI, SCL-90 MLPC	cognitive treatment more effective than relaxation training in changing use of coping strategies and pain appraisals
ter Kuile et al (1995)b	156 headache patients	longitudinal	CSQ	HI, SCL-90 MLPC	pain reduction following autogenic training and cognitive self-hypnosis training not predicted by any variable
Turner et al (1995)	139 TMD patients	longitudinal	CSQ, VPMI	pain VAS, SCL-90	modest association of pre-treatment to 3- month follow-up changes in beliefs and coping with patient improvement
Affleck et al (1999)	147 arthritis patients	longitudinal	Stone and Neale's	POMS-B, pain diary	women used more emotion-focused coping strategies than did men, men were more likely to report negative mood following pain. Diagnosis (rheumatoid or osteo-arthritis) influenced efficacy of coping strategies

NB. See Key to Abbreviations (p. 261)

Table 5.II. Studies that have examined the relationship between coping strategies and acute pain:

Author	Sample	Design	Measures Coping	Experience	Results
Rokke & al'Absi (1992)	(cold pressor)	matching / mis-matching	CCSI	pain report	CCSI valid and useful tool for selection of coping strategy to manage acute pain
Buckelew et al (1992)	50 subjects (electro-diagnosis)	correlational	CSQ-S	pain VAS STAI	catastrophising, diverting attention, self- statement positively correlated with pain, reinterpreting negative correlate
Edwards et al (1999)	46 dental patients with pulpitis; 33 controls	correlational	CSQ	pain VAS, VRI, STAI, POMS-Bi	acute pain patients catastrophised more than controls
Kahikar-Zuck et al (1997)	125 mammo-graphy subjects	longitudinal	CSQ	VAS, MPQ	women who rated their ability to decrease pain as high, experienced less pain during procedure
Baume et al (1995)	42 female patients undergoing repeated periodontal surgery	longitudinal	Stone & Neale's	behavioural (rated by dentist)	pre-surgical reports of avoidance-coping strategies related to stress behaviour; post-surgical attention-coping related to less pain

Author	Sample	Design	Measures Coping	Experience	Results
Miro & Raich (1999)	92 hyster-ectomy / oophor-ectomy patients	longitudinal	Stone & Neale's	Miller Behavioral Style Scale	no differences between coping style groups but decreased post-operative pain and disability following relaxation training

NB. See Key to Abbreviations (p. 261)

5.V. CONCLUSIONS

A large body of research on coping in chronic pain patients has demonstrated fairly consistent evidence of the association of adaptive coping strategies with better adjustment, and of maladaptive strategies with worse adjustment. Catastrophising in particular has emerged as a negative correlate with adjustment although there is some debate over whether catastrophising is a coping strategy or an aspect of distress.

Variation across pain groups has been noted and prevents general conclusions regarding coping in chronic pain, but, rather, encourages further investigation of coping strategies in specific pain conditions.

Finally, the relationship between changing coping strategies and development of chronicity of pain symptoms is one which may provide useful insight into the aetiology of chronic pain.

Chapter 6. BELIEFS IN CHRONIC PAIN

6.I. INTRODUCTION

In addition to coping strategies, beliefs held by chronic pain patients have been examined from several different but related viewpoints, principally: perception of control over personal health; perception of control over personal pain; meaning attributed to illness; meaning attributed to pain; self-efficacy; cognitive orientation; and outcome expectancy.

Researchers have developed and used measurement scales to assess patients' beliefs and to investigate the relationships of those beliefs with adjustment to, and treatment of, their pain symptoms. Approaches to assessment of beliefs have been adopted from different theoretical models.

6.II. THEORETICAL CONCEPTS

6.II.i. Locus of Control

The concept of *locus of control* originates from Rotter (1966), who distinguished beliefs in personal responsibility for events (internal locus of control) from beliefs in other responsible individuals or factors beyond personal control (external locus). Rotter considered locus of control as a personality dimension.

The external locus has subsequently been subdivided into beliefs that events are controlled by 'chance' or by 'powerful others' (Levenson & Miller, 1976).

6.II.ii. **Self-Efficacy**

The *self-efficacy* model of Bandura (1974) involves a set of beliefs held by an individual that he/she will be able to perform certain behaviours within a particular environment, as opposed to a personality trait. In other words, self-efficacy is a measure of an individual's confidence in his/her abilities with regard to a specific goal.

6.II.iii. **Illness Perceptions**

The theoretical framework is the self-regulation model of Leventhal and colleagues, who have proposed that patients' illness representations are based around distinct components which, in turn, determine coping (Leventhal et al, 1984; Leventhal & Diefenbach, 1991). Thus they maintain that each patient will have their own ideas about the identity, cause, time-line, and consequences of their illness. Lau and colleagues have indicated that patients' models also incorporate beliefs about the cure and controllability of the condition. Recent overviews in this area, based on differing methodologies across a range of different clinical conditions, confirm the consistency and validity of these five components of patients' illness representations (i.e. identity, cause, time-line, consequence, control/cure; Skelton & Croyle, 1991).

6.II.iv. **Cognitive Orientation**

The theory of cognitive orientation (Kreitler & Kreitler, 1976) involves "assignment of meaning to inputs, evocation of beliefs to explore the meaning of behavioural alternatives, belief clustering to determine behavioural intent orienting toward a

given course of action, and a behavioural program to enable the carrying out of a behavioural intent" (Kreitler & Kreitler, 1987, p.324).

It will be seen that the above concepts of beliefs and appraisals, together with outcome expectancies, are applicable to the experience of chronic pain.

6.III. MEASUREMENT SCALES

6.III.i. **The Multidimensional Health Locus of Control scale (MHLC)** (Wallston et al, 1978)

This is an 18-item, 6-point Likert scale measuring the orientation of subjects' health locus of control beliefs, including Internal, Powerful Others (such as health care professionals) and Chance factors. The three subscales are internally consistent with alpha reliabilities ranging from 0.67 to 0.77 (Wallston et al, 1978). The authors encouraged adaptation of the scale for specific populations. This has been done for chronic pain populations by, for example, simply substituting the word 'pain' for 'health' (Crisson & Keefe, 1988).

6.III.ii. **The Illness Attitudes Scale** (Kellner, 1986)

This scale is designed to assess fears, beliefs and attitudes associated with hypochondriasis and abnormal illness behaviour. Although these concepts are prejudicial in the presumption that there is indeed a 'normal' illness behaviour, the scale has been proposed as a useful tool in chronic pain, particularly for the assessment of pain-related fear (Hadjistavropoulos & Asmundson, 1998).

6.III.iii. **The Survey of Pain Attitudes (SOPA)** (Jensen et al, 1987)

This is a 24 true-false item scale measuring subjects' beliefs in 'medical cure', 'pain control', 'solicitude', 'disability' and 'medication'. Revisions of the instrument added two more subscales of 'emotion' and 'harm' (Jensen et al, 1994b), resulting in a 57-item, 5-point Likert scale with internal consistency of 0.71 to 0.81.

In a sample of multidisciplinary pain centre patients, Jensen et al (1999) found pain beliefs (specifically: the belief that pain signals alarm; that one is disabled; and that solicitous response from others are appropriate - SOPA) predictive of changes in observed pain behaviours, whether these behaviours were reported by the patient, his/her spouse, or an independent observer.

6.III.iv. **The Meaning of Illness Questionnaire (MIQ)** (McAdams et al, 1989)

This is a 33-item 7-point Likert scale measuring 5 factors: 'impact of illness', 'type of stress (negative attitude of illness as a harm, loss, threat and function context)', 'degree of stress (change in commitments, secondary appraisal of coping resources)', 'positive attitude (challenge, hope, motivation, control)', and 'expectancy / recurrence'. The MIQ has a strong relationship to health outcomes (Browne et al, 1990).

6.III.v. **The Pain Beliefs and Perceptions Inventory (PBAPI / PBPI)** (Williams & Thorn, 1989)

This is a 16-item, 4-point Likert scale measuring subjects' beliefs in 'pain stability', 'pain as a mystery' and 'self-blame'. The 3-factor solution was subsequently changed to a 4-factor solution, splitting the 'time' (pain stability) factor into 'permanence' and

'constancy' after two studies failed to replicate the original structure (Strong et al, 1992; Herda et al, 1994). This alteration apparently allowed for "greater interpretation of which type of temporal beliefs were associated with increased pain ratings" (Williams et al, 1994, p.76).

6.III.vi. **The Pain Beliefs Questionnaire (PBQ)** (Edwards et al, 1992)

This is a 12-item scale, response involving endorsement of one of a choice of six qualifying adverbs ('never' to 'always'), designed to assess beliefs about the experience of pain, its causes, consequences, and factors influencing its severity. The authors identified two subscales: 'organic beliefs' and 'psychological beliefs'; with internal consistency of 0.71 and 0.73 for chronic pain patients.

6.III.vii. **Chronic Pain Self-Efficacy Scale** (Anderson et al, 1995)

This scale has been developed in an outpatient group, and has three subscales: pain management, coping with symptoms, and physical function, confirmed in a replication sample. Subscale scores correlated significantly with measures of depression, hopelessness, somatic preoccupation, and adaptation to chronic pain. Amstein et al (1999) used path analysis methodology to demonstrate the role of self-efficacy as a mediator between pain and disability. In a sample of specialist pain clinic patients, self-efficacy increased the prediction of the variance in disability (Pain Disability Index, PDI: Pollard, 1985) by 12 %. Pain intensity (VAS) alone predicted 32% of the variance.

6.III.viii. **The Illness Perceptions Questionnaire (IPQ)** (Weinman et al, 1996)

This scale was developed to assess patients' representations of illness in order to improve the understanding of illness-related coping and to develop interventions to facilitate self-management in chronic illness. The IPQ is a theoretically derived measure comprising five subscales: *identity* - the symptoms the patient associates with the illness; *cause* - personal ideas about aetiology; *consequences* - expected effects and outcome; and *control / cure* - how one controls or recovers from the illness.

6.IV. FINDINGS

Table 6.I. describes pertinent studies in this field. Abbreviations are explained in the key (p. 261).

6.IV.i. **Locus of Control**

Chronic pain patients endorsing internal locus of control are better adjusted to living with pain in terms of psychological distress than those endorsing chance or powerful others as responsible (Crisson & Keefe, 1988; Toomey et al, 1991). One study also found 'internal' patients to report less pain intensity (Toomey et al, 1991). Buckelew et al (1990) found no such relationships, however.

In temporomandibular pain patients, locus of control was found to be more 'external' than in healthy controls (Marbach et al, 1988) but failed to predict treatment completion (Funch & Gale, 1985), although completion of treatment does not necessarily imply response to treatment.

6.IV.ii. **Pain Beliefs**

Internal locus of control is related to beliefs in pain as enduring and mysterious, which are associated with greater pain report, less perceived ability to control pain by coping, more 'passive' coping strategies, greater psychological distress, poorer treatment compliance and less improvement after treatment (Williams & Thorn, 1989; Williams & Keefe, 1991; Herda et al, 1994; Williams et al, 1994).

For patients with low pain levels, perceived control over pain is associated with more physical activity, even after controlling for coping strategies (Jensen & Karoly, 1991). Patients showing the greatest improvement after treatment (acupuncture) hold more beliefs orienting toward pain relief pre-treatment (Kreitler & Kreitler, 1987). Changes in pain-related beliefs and coping strategies are associated with improvement after multidisciplinary treatment (Jensen et al, 1994).

6.IV.iii. **Self-Efficacy and Expectancy**

High self-efficacy patients show less pain and greater improvement with treatment both subjectively and objectively (Kores et al, 1990; Buckelew et al, 1994; Anderson et al, 1995). Council et al (1988) argue that pain response expectancies influence performance and associated pain through their effects on efficacy expectancies. Return-to-work expectation is associated with completion of a work rehabilitation program (Carosella et al, 1994).

6.IV.iv. **Private Body Consciousness**

Private Body Consciousness (PBC), or attentional self-focus, is another construct purported to explain some of the variance in pain-related anxiety. Pain patients

reporting high PBC (Body Consciousness Questionnaire, BCQ: Miller et al, 1981) reported more pain (Pain Mannequin: Ahles et al, 1987) but did not differ on anxiety (MMPI) (Ferguson & Ahles, 1998). PBC did not differ between pain patients and controls and was considered a dispositional amplifier of aversive stimulation.

Table 6.I. Studies of Beliefs in Chronic Pain

Author	Sample	Design	Measures Beliefs	Other	Results
Funch & Gale (1985)	78 TMJ pain patients	correlational	HLC	treatment completionPDQ, TMAI	no predictive value
Marbach et al (1988)	151 TMPDS patients	correlational	RLOC, LLOC	versus controls	cases show higher external l.o.c. perception (RLOC only)
Crisson & Keefe (1988)	62 chronic pain patients (mostly back)	correlational	MHLC - modified	CSQ, SCL-90-R	'chance' l.o.c. correlated with psychological distress and with 'passive' coping
Buckelew et al (1990)	161 chronic pain patients (back, leg, etc.)	correlational	MHLC	WCQ, SCL-90-R, pain (sites, intensity), disability rating	no correlation between l.o.c. and pain/distress; males in l.o.c. clusters differ only in age (internals younger), females in educational level (internals higher)
Toomey et al (1991)	51 chronic pain patients (mostly myo-fascial)	correlational	MHLC (modified: PLOC)	pain intensity and frequency, health care use, functional interfer-ence (VAS's)	internal l.o.c. negatively correlated with pain intensity and frequency
Harkapaa et al (1996)	76 low back pain patients	correlational	MHLC, GLOC, BPLC	LBP index, GHQ, PRSS, PRCS, behavioural activity score	Internal control beliefs associated with coping self-statements; external control beliefs with catastrophising and hopelessness

Author	Sample	Design	Measures Beliefs	Other	Results
Williams & Thom (1989)	87 industrially injured chronic pain patients	correlational	PBAPI, MHLC	pain intensity (Likert), treatment compli-ance, RSES, MMPI	belief in pain endurance associated with greater pain intensity, decreased treatment compliance (health psychology and physical therapy); belief in pain as mysterious associated with little improvement. Both beliefs associated with internal l.o.c.
Williams & Keefe (1991)	120 chronic pain patients (mostly back)	correlational	PBAPI	MPQ-PRI, CSQ	beliefs in pain as enduring and mysterious associated with perceived reduced ability to control pain by coping strategies
Herda et al (1994)	193 chronic pain patients (back, musculoskeletal)	correlational	PBAPI (German version)	pain intensity (VAS) and frequency, TL, STAI, PRSSS	belief in pain as mysterious associated with trait-anxiety and catastrophising
Williams et al (1994): (Study 3)	37 chronic pain patients (back, head, neck, etc.)	correlational	PBPI, re-scored (=PBAPI)	MPQ-SF, BAI, BDI, NEO-PI, ADS	beliefs in mystery, pain permanence and self-blame associated with neurotic traits; belief in pain as mysterious correlated with BAI, BDI; belief in pain as constant associated with ADS
Williams et al (1994): (Study 4)	148 chronic pain patients (mostly back)	correlational	PBPI	CSQ	beliefs in mystery, pain permanence and self-blame associated with less praying/hoping and more catastrophising

Author	Sample	Design	Measures Beliefs	Other	Results
Jensen & Karoly (1991)	118 chronic pain patients (back, head, etc.)	correlational	SOPA	pain intensity (0-100) and frequency, CSQ, CES-D, SWLS, HAQ, MPI, health care use, medication use	perceived control over pain associated with better psychological functioning, mediated by coping strategies; perceived control associated with greater activity(low pain only), even after controlling for coping
Jensen et al (1994a)	94 chronic pain patients (back, head, leg, etc.)	longitudinal	SOPA	CSQ, BDI, SIP, health care use	changes in pain-related beliefs and coping strategies associated with improvement after multidisciplinary treatment
Jensen et al (1999)	121 chronic pain patients and spouses / partners	correlational	SOPA	SIP, PBCL (Pain Behavior Check List, Kems et al, 1991)	harm beliefs (hurt indicates damage and activity should be avoided) closely linked to functioning and pain behaviour
Turner et al (2000)	169 pain clinic patients	correlational	SOPA, PBPI	VAS, Roland Scale, CES-D, CSQ, CPCI	belief scores independently predicted physical disability and depression.
Slater et al (1991)	31 male chronic low back pain patients, 19 healthy controls	correlational	PAIRS	pain intensity (VAS), SIP, WPII, SPS, CEQ, BDI	PAIRS found to be a valid measure of impairment without being related to pain
Edwards et al (1992)	40 rheum-atology patients	correlational	PBQ, MHLC	pain intensity (VAS) and duration, medication	'chance' and 'powerful others' scales of MHLC correlated with PBQ 'organic beliefs' scale, 'internality' (MHLC) correlated with 'psychological beliefs' (PBQ)

Author	Sample	Design	Measures Beliefs	Other	Results
Kores et al (1990)	34 chronic pain patients (mostly back)	correlational	modified self-efficacy scale, HLC	MPQ, SHI, BDI, short- (exercise) and long-term (Rating of Improve-ment scale) outcome	high self-efficacy subjects pre-treatment showed and reported greater improvement post-treatment
Dolce et al (1986)	14 chronic pain patients (12 back)	longitudinal	self-efficacy ratings	exercise tolerance, worry / concern ratings	self-efficacy expectancies and exercise tolerance increased across treatment, worry/concern decreased
Jensen et al (1991)	114 chronic pain patients (back, leg, head, etc.)	correlational	self-efficacy and outcome expect-ancy ratings	use of coping strategies, pain intensity (0-100), SIP	beliefs re. capabilities related to reported coping efforts; beliefs re. consequences of coping efforts unrelated to coping
Buckelew et al (1994)	73 fibro-myalgia patients	correlational	Arthritis Self-efficacy Scale	pain VAS, CES-D, pain behaviour observat-ion	high self-efficacy patients exhibited fewer pain behaviours; depression was unrelated to pain behaviour
Anderson et al (1995)	141 chronic pain patients (mostly back)	correlational	CPSS	BDI, BHS, BPPA, MPI	CPSS found to be a valid measure of self-efficacy beliefs of individuals with chronic pain, negatively correlating with pain, depression and hopelessness

Author	Sample	Design	Measures Beliefs	Other	Results
Marlowe (1998)	120 headache sufferers	longitudinal	unvalid-ated self-efficacy scale	diary of headaches and stressful events	relationship between stressful events and headache strongest for those with low self-efficacy, and weakened as self-efficacy increased
Arnstein et al (1999)	126 chronic pain clinic patients	correlational	CPSS	VAS, PDI, CES-D	self-efficacy mediates the relationship between pain and disability
Lefebvre et al (1999)	128 rheumatoid arthritis patients	longitudinal	ASES	pain diary, POMS-Bi, CSQ, Stone & Neale's daily coping inventory	self-efficacy significantly related to pain, mood, coping and coping efficacy
Kreitler et al (1987)	30 chronic pain patients (multiple sites)	correlational	Cognitive Orientat-ion questionn-aire	pain character-istics, patient and physician ratings	highly improved patients post-treatment (acupuncture) held more beliefs orienting toward pain relief pre-treatment
Weir et al (1994)	221 chronic pain patients	correlational	MIQ	PAIS-SR, BMSS, pain and psycho-social progress (GP's ratings)	psychosocial adjustment was best explained by social support which strongly correlated with the cognitive variables of meaning
Bates et al (1993)	372 multi-ethnic chronic pain patients	correlational	EPQ	MPQ-PRIT	l.o.c. style related to ethnic identity
Council et al (1988)	40 chronic pain patients (mostly back)	correlational	MAPPS	observed move-ments, SIP, daily activity diary	self-efficacy and pain response expectancies correlated with actual performance

Author	Sample	Design	Measures Beliefs	Other	Results
Carosella et al (1994)	168 low back pain patients	correlational	return to work expectat-ion (VAS)	pain intensity (VAS), WCDI, MCMI-II, WES	Early Discharge group reported lower expectation to return to work than rehabilitation completers
Ferguson & Ahles (1998)	144 pain clinic patients and 31 controls	correlational	BCQ	pain mannequin, MMPI	PBC related to pain intensity in patient group; no inter-group difference in PBC
Moss-Morris et al (1996)	233 Chronic Fatigue Syndrome sufferers	correlational	IPQ	COPE, MHI-5, SIP	illness perceptions explained a greater percentage of the variance in disability and distress than did coping strategies. Those with a strong illness identity, who believed their illness to be out of their control, caused by stress, and of serious consequence, were most disabled and distressed.

6.V. METHODOLOGICAL PROBLEMS

As with any self-report scales, there is the possibility of social desirability bias in these studies, though this is partly controlled with the use of the Marlowe-Crowne Social Desirability Scale (for example: Jensen & Karoly, 1991).

Also, selection of samples varies from rehabilitation programmes and outpatient departments to specialist pain clinics, which makes generalisation of findings difficult.

The sheer number and variety of measurement scales obviously makes summary of findings in this field problematic.

6.VI. CONCLUSIONS

The conceptually related ideas of locus of control, pain beliefs, self-efficacy and expectancy have associations with coping and adjustment in chronic pain. In addition, pain beliefs are associated with physical disability and depression, independently of coping and catastrophsing (Turner et al, 2000). It therefore seems appropriate to assess these beliefs as part of the clinical management of patients. With regard to temporomandibular pain, given its unknown aetiology, patients' beliefs would be of particular interest.

The Pain Beliefs Questionnaire (PBQ, Edwards et al, 1992), rather than focusing on patients' beliefs in their ability to control pain, is intended to assess beliefs about the causes of pain and will be illuminating in TMD, particularly in comparison with a pain condition of known 'organic' aetiology, namely post-extraction pain following third molar removal. The Illness Perceptions Questionnaire (Weinman et al, 1996) also considers consequence, cure and time-line, and will therefore be of interest in

the later comparison of TMD with headache, which, whilst similarly idiopathic, is of established and significant impact on quality of life.

Chapter 7. DEPRESSION IN CHRONIC PAIN

7.I. INTRODUCTION

The prevalence of depressed affect in chronic pain populations is 10 - 100% (Romano & Turner, 1985). The size of this range is due to several methodological problems inherent in assessment studies. Prevalence of depressive symptoms in chronic pain groups is, however, consistently higher than in the general population and many other medical populations (Romano & Turner, 1985).

The psychiatric classification criteria for depression are syndromal (for example: DSM- IV, Diagnostic and Statistical Manual of Mental Disorders, American Psychiatric Association, 1994). Diagnoses are defined as syndromes, comprising combinations of affective, cognitive and somatic-vegetative signs and symptoms of depression. In addition, a certain duration and other inclusion and exclusion criteria are specified (Estlander, 1996).

Whilst there is little evidence for a greater prevalence of syndromal depression in chronic pain patients, relative to the general population; *depressive symptoms* are more common (Romano & Turner, 1985). Depressive symptoms may be quantified using self-report scales and it is therefore appropriate to assess negative affect in pain populations by means of questionnaire.

7.II. RELATIONSHIP BETWEEN PAIN AND DEPRESSION

Brown (1990) discusses three principal theories of the pain-depression relation:

7.II.i. Depression evokes chronic pain by increasing pain sensitivity and reducing pain tolerance thresholds.

Pain reflects a masked depression state in a pain-prone individual (Blumer & Heilbronn, 1982). There would appear to be no empirical support for traditional psychodynamic models of chronic pain as a conversion reaction, a pain-prone disorder or masked depression, yet there is some evidence for predisposing factors: there is a significant positive relationship between depression and history of childhood sexual and physical abuse in chronic pain patients (Goldberg, 1994).

7.II.ii. Depression occurs as a secondary reaction to chronic pain.

Chronic pain may produce an incapacitating physical condition and depression develops as a function of the sustained reduction in physical and social activities (for example: Hendler, 1984). The operant approach suggests that this reduction in activities in turn leads to a reduction in positive reinforcement and thereby depression. Indeed, correlations with depression are stronger for interference with activities (Haythornthwaite et al, 1991; von Korff & Simon, 1996), physical impairment (Rudy et al, 1988), life-control (Turk et al, 1995) and loss of paid work (Fifield et al, 1991), than are correlations between depression and pain report itself (Estlander, 1996).

7.II.iii. **Chronic pain and depression occur simultaneously because of a similar psychological or biological origin.**

For example: 5HT deficiency or impaired tyramine conjugation (Aghabeigi et al, 1993). The clinical efficacy of anti-depressant drugs, in double-blind trials versus placebo in the management of chronic pain, has also been cited as evidence for this last theory (for example: Johansson & von Knorring, 1979; Hameroff et al, 1982; Feinmann & Harris, 1984).

7.II.iv. **Problems with Theories**

Some patients with chronic pain are depressed whilst others are not, just as some patients with psychiatric disturbance have chronic pain whilst others do not. The above theories fail to explain these observations.

Cognitive theories describe how depression develops secondary to cognitive distortions, negative beliefs and attributions, and dysfunctional attitudes (for example: Beck, 1976). Studies of chronic pain patients have shown some support for the significance of cognitive factors in depression (Rodin et al, 1991; Turk & Rudy, 1992) and have demonstrated the differences in cognitions between depressed and non-depressed patients (Lefebvre et al, 1981; Holzberg et al, 1993; Smith et al, 1994). The relative individual roles of these factors remain unclear.

A new "diathesis-stress" framework has been proposed (Banks & Kerns, 1996) to explain the high comorbidity between chronic pain and depression. This approach encourages identification of vulnerability factors in the individual (diathesis) as well as investigation into the nature of the stressor

Sullivan et al (1992) remarked that: "examination of the current literature on chronic pain and depression reveals a conceptually fragmented area with little cross-study consistencies in theoretical framework, methodology, or findings" (p.5).

7.III. METHODOLOGICAL PROBLEMS

7.III.i. Disparate Assessment Methods

Most studies have relied on self-report measures of depression, principally the Beck Depression Inventory (BDI, Beck et al, 1961) and the Center for Epidemiology Studies Depression Scale (CES-D, Radloff, 1977). A few studies have used standardised psychiatric interview (DSM III, for example: Fishbain et al, 1986). Other studies have used unspecified criteria. Similarly, the method of pain assessment has not always been specified (Romano & Turner, 1985). Thus, an overview requires comparison of relations determined between different methods of mood assessment and different measures of pain assessment.

7.III.ii. Selected Subject Groups

Studies of pain patients have included samples drawn from pain clinics, private practice, psychiatric consultation-liaison services and psychiatric inpatient wards (Romano & Turner, 1985). These patients are probably not representative of chronic pain populations in general, partly because of a neurotic bias in any specialised clinical population (Merskey et al, 1985). However, any sample is likely to incur some form of bias and, in fact, that bias may be fundamental in determining whether an individual enters a patient group, i.e. treatment-seeking bias.

7.III.iii. **Overlap in Symptoms of Pain and Depression**

A recent criticism leveled against the assessment of depression in chronic pain groups is that depression ratings are elevated by the confounding of symptoms of depression and chronic pain (for example: Wesley et al, 1991; Williams & Richardson, 1993). Specifically, the "somatic" items of sleep disturbance, change of appetite, fatigability and loss of libido are considered to reflect pain rather than depression (Estlander, 1996). The problem remains of how to avoid this confounding of somatic symptoms on self-report scales. Removal of these items might result in underestimation of depression in pain patients (Sullivan et al, 1992) and a compromise of separate scoring of somatic items might be preferable (Williams & Richardson, 1993). The Hospital Anxiety and Depression scale (HAD, Zigmond & Snaith, 1983) has been designed to avoid confounding of symptoms. The high level of agreement between the depresssion scale of the HAD and the BDI in most chronic pain groups indicates that endorsement of somatic items in the BDI does reflect depression rather than pain (Miles et al, 1995).

7.III.iv. **Lack of Prospective Studies**

The majority of studies of depression in chronic pain groups have been correlational. These have provided no insight into the causal relationship between pain and depression and therefore no preferential evidence for either of the first two theories above. More recently, longitudinal studies have aimed to address this.

7.IV. RECENT LONGITUDINAL STUDIES

Three studies have examined the temporal relationship between pain and depression. Brown (1990) invited 744 rheumatoid arthritis patients (diagnosed by practicing rheumatologists) to participate in a prospective study by post. 243 patients (75% females) completed self-report measures of pain (Arthritis Impact Measurement Scale and Visual Analogue Scale) and depression (CES-D) on a six-monthly basis. A structural modeling technique was used to infer a causal relationship between the two variables. Data from the first 12 months did not support a causal relationship. However, data from the last 12 months of the study strongly supported pain as a predictor of exacerbation of depression over a 6-month period, even when controlling for prior depression.

Magni et al (1994) used data collected in two surveys of the general population of the United States, seven years apart, on a sample of 2324 participants. Pain assessment was by self-report, depression by self-report using the CES-D. Although the data supported both directions, the odds ratio for pain predicting depression was notably larger than that for depression predicting pain. These two studies therefore provide some support for depression as a sequela of chronic pain.

In a further examination of the 'chicken-or-egg' debate regarding pain and depression, Dohrenwend et al (1999) interviewed probands of 106 myofascial face pain patients and 118 non-pain acquaintance controls (all female), and a random sample of one adult first degree relative for each. Although facial pain patients were recruited from one specialist clinic and diagnostic criteria predated the Research Diagnostic Criteria (RDC/TMD: Dworkin & LeResche, 1992), psychiatric interview for MDD involved the Structured Clinical Interview for DSM-III. The results

showed elevated MDD and Depressive Spectrum Disorders (DSD) in the first-degree relatives of control probands with early-onset MDD, but no such elevations in relatives of pain patients. This finding is consistent with the hypothesis that "living with chronic myofascial face pain contributes to elevated rates of depression" and inconsistent with the alternative theory that pain is a variant of depression.

7.V. DEPRESSION AND KNOWN CAUSE OF PAIN

Studies comparing subjects with organic lesions and subjects with indeterminate chronic pain, report considerably higher prevalence of depression among the latter (Magni, 1987). This suggests that beliefs and attributions about the cause of their symptoms may affect psychological well-being in chronic pain patients - something of particular interest in an idiopathic pain condition such as TMD.

7.VI. CONCLUSIONS

7.VI.i. Valid diagnosis of major depressive disorder (MDD) can only be made on the basis of structured clinical psychiatric interview based on the Research Diagnostic Criteria (RDC, Spitzer et al, 1978) or DSM-IV, by which MDD is defined. However, self-report questionnaires (BDI, CES-D, HAD) will yield a numeric rating of depressive symptoms, or, in other words, a measure of negative affect. The potential confounding of "somatic" symptoms in chronic pain patients must be recognised.

7.VI.ii. In assessing comorbidity of pain and depression, subjects are best selected from general population or primary care settings rather than specialist pain clinics, early in the natural history of the pain condition (von Korff & Simon, 1996).

7.VI.iii. Several theories of the pain-depression relation have been proposed, deriving from different psychological schools, but the evidence to support any one theory is scant. The direction of the causal relationship between pain and depression will only be elucidated through well-designed prospective studies, and there is some recent evidence for depression as a sequela of pain, or even a sequela of disability (Pincus & Williams, 1999).

7.VI.iv. The "diathesis-stress" approach encourages identification of vulnerability factors in the individual as well as investigation into the nature of the stressor, and provides a useful theoretical basis from which to advance the study of depression in chronic pain.

Depression is not, however, the only mood state that feeds into the pain experience. Chapter 3 has shown the importance of anxiety in the acute pain experience, and anxious mood has also been investigated in chronic pain.

Chapter 8. ANXIETY IN CHRONIC PAIN

8.I. INTRODUCTION

Anxiety in chronic pain patients has received far less attention than has depression. This is perhaps surprising given that chronic pain syndromes often involve unpredictable recurrences of pain. Idiopathic pain might therefore produce anxiety both through the lack of predictability of recurrence and the uncertainty of cause and outcome.

The muscle tension hypothesis, referred to in the introduction, proposes that anxious people endure more tension than others, which, in turn, causes muscle tightening. This muscle tightening eventually becomes a source of pain leading to additional anxiety, and thus perpetuating the anxiety / pain relationship (Merskey, 1980). This might be of particular significance in myalgia syndromes, such as TMD. Unfortunately, there is little evidence to support this attractive hypothesis.

Studies of experimental acute pain in humans have shown that pain-related anxiety increases pain perception whilst pain-irrelevant anxiety decreases pain perception, both being mediated by attention (Janssen & Arntz, 1996). Chronic pain patients might thus be expected to report increased pain intensity if anxious about their pain.

8.II. LITERATURE REVIEW

Table 8.I. summarises pertinent studies.

8.III. FINDINGS

8.III.i. Anxiety and Pain Symptoms

In a group of fibromyalgia patients, Kurtze et al (1998) found independent additive effects of anxiety and depression upon fatigue (Symptom Checklist, SCL) and pain (Ursin Health Inventory, UHI: Ursin et al, 1988). Subjects with high anxiety and low depression did not suffer from *widespread* pain, this being a diagnostic criterion for fully developed fibromyalgia syndrome, suggesting that high anxiety is associated with an 'over-reaction' to *local* pain. The authors cited the Muscle Tension Hypothesis as a possible mediator for this discrepancy, yet, as noted above, there is no physiological evidence for this theory. Although anxiety appeared unrelated to duration in this study, the association of high anxiety with a local and potentially, relatively responsive pain condition might provide an explanation for the prevalence of greater anxiety in acute TMD populations than that in chronic populations (Gatchel et al, 1996), since the most anxious and less depressed individuals, in responding to treatment, would not be present in a more chronic group.

8.III.ii. Anxiety and Other Symptoms

Physiological symptoms of pain-related anxiety (PASS, eg. palmar sweating) were found to be a stronger predictor of non-specific physical complaints (Modified Somatic Perception Questionnaire, MSPQ: Main, 1983) in chronic pain patients than were cognitive and somatic depression (BDI) (McCracken et al, 1998). More frequent non-specific physical complaints were also found to predict greater disability (SIP). The findings, though from correlational data, were taken to support a model in which non-specific physical complaints are the direct result of distress due

to the pain experience, and that, once again, distress is a consequence rather than a cause of chronic pain.

8.III.iii. **Anxiety Sensitivity**

A related concept, anxiety sensitivity (AS), the tendency to become fearful, was found to be high in recurrent headache patients, relative to other chronic pain groups, and those highest in AS reported more adverse effects related to their pain, specifically: greater depression, anxiety, pain-related escape/avoidance behaviour and fear of pain (Asmundson et al, 1999). These relationships have also been found in musculoskeletal pain groups.

Table 8.I. Studies that have investigated anxiety in chronic pain patients:

Author	Sample	Design	Measures Anxiety	Other	Results
Krishnan et al (1985)	71 low back pain patients	correlational	HAS	RCD-D	symptoms of anxiety more common in depressed patients
Dworkin et al (1992)	19 acute Herpes zoster patients	longitudinal	STAI	MPQ, BDI, LSES, DAS, ASQ, IBQ, SRE	patients who developed chronic pain had eg. initially higher trait and state anxiety
McCrack-en et al (1992)	104 chronic pain patients (mostly back)	correlational	PASS, STAI, CSAQ	BDI, CSQ, MPQ, MPI, PDI	PASS a valid and reliable measure of pain-related fear and anxiety, which are associated with disability and interference due to pain
McCrack-en et al (1993)	43 low back pain patients	longitudinal	PASS	pain prediction during SLR test, pain and anxiety experience (0-100)	high pain-anxious S's reported greater experienced anxiety; low pain-anxiety S's underpredicted pain
Williams & Richard-son (1993)	207 chronic pain patients (mostly back)	correlational	STAI	BDI, PCQ, PSEQ	anxiety associated with 'sadness about health' and 'self-reproach' factors of BDI (but not with 'somatic disturbance' factor)
Williams et al (1994)	37 chronic pain patients (various)	correlational	BAI	PBPI, MPQ, BDI, NEO-PI, ADS	anxiety associated with belief in pain as mysterious and permanent
Casten et al (1995)	479 geriatric institution-alised S's	correlational	DSM-III-R, POMS	pain intensity and frequency (1-5)	anxiety/pain and depression/pain relationships exist even when controlling for strong correlation between anxiety and depression

Author	Sample	Design	Measures Anxiety	Other	Results
Morley & Pallin (1995)	100 chronic pain patients (CLBP, RA, headache and controls)	correlational	HAD	MPQ, CSQ, verbal descriptor similarity judgement task	anxiety differed between groups: CLBP > headache > RA. > controls
Novy et al (1995)	251 chronic pain patients	correlational	STAI	BDI, BHS, MMPI, PSPI	'negative attitudes/suicide' and 'performance difficulty' factors of BDI correlated strongly with eg. anxiety ('physiological manifestations' factor only weakly)
Gatchel et al (1996)	51 acute & 50 chronic TMD patients	case control	SCID (DSM-III-R)		high rate of anxiety disorders in acute group (TMD <6 months) (47 – 53 %) compared with chronic group.
Mc-Cracken et al (1998)	210 pain clinic patients	correlational	PASS, MSPQ	pain VAS, BDI, SIP	anxiety and depression were significant 'predictors' of physical complaints, but physiological symptoms of pain-related anxiety were stronger 'predictors'.

NB. See Key to Abbreviations (p. 261)

8.IV. DISCUSSION

Once again, the wide variety of assessment scales makes comparison of studies difficult. Sample selection also varies from specialist pain clinics to outpatient departments and a care facility for the elderly. As with assessment of depression in chronic pain patients, there remains the problem of somatic symptom confounding (insomnia, for example) and over-estimation of anxiety.

The great majority of studies relies on a correlational design and consequently provides no information about the direction of the anxiety / pain relationship over time. Of the two longitudinal studies cited, Dworkin et al's study (1992) is weakened by the effect of pre-existing pain on the assessment of variables including anxiety; whilst the predictions based on initial pain-anxiety in McCracken et al's study (1993) do not include pain intensity *per se*.

8.V. CONCLUSIONS

Despite methodological problems, there is evidence of greater prevalence of anxious symptoms in chronic pain populations than in controls (eg. Krishnan et al, 1985). However, there is a lack of prospective studies to elucidate the causal direction of the anxiety / pain relationship.

Since there is compelling evidence of normal personality structure in chronic pain patients (eg. Wade et al, 1992; Schnurr et al, 1990), it seems likely that anxiety develops as a sequela to pain but then contributes to its endurance and impact.

Assessment of anxiety in chronic pain patients, in addition to depression, using valid and reliable measures, is important.

Chapter 9. STUDIES: AIMS, MEASURES AND HYPOTHESES

9.I. INTRODUCTION

9.I.i. Cognitive Factors in TMD

Psychological approaches in facial pain have historically focused on two aims: investigation of an underlying psychological defect to demonstrate a psychosomatic mechanism; and management with psychotropic agents. Neither of these approaches has been particularly successful: there is little evidence for a TMD-prone personality (Schnurr et al, 1990); and antidepressant medication is largely equivocal in efficacy to other modes of treatment such as splint therapy (Tversky et al, 1991).

More recently, cognitive behavioural therapy has been advocated (Turner et al, 1995; Turk et al, 1996; Harrison et al, 1997), and has shown some modest benefit. However, little thought has been devoted to establishing which cognitions and behaviours are of significance in TMD, which may be adaptive and which maladaptive, which may be encouraged and which discouraged, in order to reduce pain, disability, and distress (Harness & Rome, 1989; Villarosa & Moss, 1985).

An attempt at identifying cognitive factors in facial pain has been made using the Rational Beliefs Inventory, a measure of rationality derived from Ellis' theory of Rational Emotive Therapy (Ellis & Bernard, 1986), a cognitve-behavioural treatment paradigm specifically focusing on maladaptive cognitions (Schwartz & Gramling, 1997). Facial pain sufferers were found to harbour several cognitive errors relative to no-pain controls. These included: *projected misfortune* (anticipation of future stress and ill fate), *guilt* (a set of self expectations and obligations that are often unrealistic and rigid), *caring and helping* (the belief that caring and helping others is required rather

than preferable), *control of emotion* (modulation of affect based on the constraints of a given situation). However, the study involved university students, not patients, and no formal diagnostic criteria and the clinical relevance of the findings is therefore highly questionable.

9.I.ii. **Comparison with Other Pain Groups**

Comparison was made with an acute pain group of patients having undergone extraction of a lower third molar (wisdom) tooth. This model of acute pain is ideal for comparison with 'chronic' TMD pain because of its bodily location and associated jaw dysfunction. It is also a model widely used in analgesic trials. Comparison of TMD with a condition of known cause, consequence and time-line (i.e. how long it will last), in terms of disability, ought to suggest how disability is influenced by these pain beliefs (i.e. cause, consequence, time-line).

Recent literature, and, in particular, that endorsing a psychosomatic genesis, has suggested that many chronic idiopathic ("functional") conditions are so similar in terms of symptoms, lack of identifiable pathology, and associated psychosocial factors, that they should be considered as one. Wessely and colleagues have postulated that: "the existence of specific somatic syndromes is largely an artefact of medical specialisation. That is to say that the differentiation of specific syndromes reflects the tendency of specialists to focus on only those symptoms pertinent to their specialty, rather than any real differences between patients" (Wessely et al, 1999: p.936).

TMD and primary headache differ in that the former is managed by General Dental Practitioners and Oral & Maxillofacial Surgeons, the latter by General Medical

Practitioners and Neurologists. The relationship with psychological stress is widely accepted for headache, particularly tension-type headache, but not for TMD. Comparison of the relative contributions of psychological factors, such as illness perceptions, to headache and TMD, ought to shed valuable light on their similarities and differences, and on the extent to which these can be attributed to the individual characters of the pain conditions. Stress, cognitions, coping and mood have been studied, and cognitive behavioural therapies assessed, in headache patients (Madland & Feinmann, 2000). The professional and lay acceptance of psychological factors as contributory is perhaps greater, particularly in tension-type headache, than in TMD. Headache patients are proposed for comparison with TMD sufferers in terms of the psychological factors described.

9.I.iii. **Pain Intensity, Character, Location, and Cognitions**

Although pain beliefs have been shown to influence chronic pain patients' psychosocial functioning, supporting a cognitive-behavioural model (Jensen et al, 1999), no study appears to have investigated the influence of pain intensity and character on those beliefs, despite the stated need "to identify for each patient the uniquely challenging or stressful aspects of his or her pain experience" (Banks & Kerns, 1996). This will be a further focus for study.

9.I.iv. **Anger Style and Alexithymia**

Whilst anxious and depressed mood have been extensively investigated in TMD, few studies have addressed *anger*. In a mixed chronic pain group, Burns et al (1998) found significant 'anger suppression x gender' effects, such that anger suppression among

males correlated negatively with treatment response, even after controlling for trait anger. No such relationship was seen in female patients. This study included a measure of anger in response to its report by TMD patients in the previous study.

Alexithymia ("no words for mood") is a multidimensional construct from psychosomatic research defined by the following cognitive-affective characteristics: difficulty in identifying feelings (Factor 1); difficulty in describing feelings (Factor 2); and externally-oriented thinking (Factor 3). Alexithymia has been found to be raised in chronic pain populations compared with controls, even after controlling for treatment-seeking bias (Lumley et al, 1997), and might provide an explanation for continuing facial pain as well as lack of success of imagery-based interventions. A Finnish birth cohort study of nearly 5000 subjects found the proportion of alexithymics (Toronto Alexithymia Scale score over 60) to be higher in subjects with the most orofacial symptoms than in asymptomatic subjects. After adjusting for depression, marital status, and self-rated health, a significant association remained between alexithymia and the symptoms mentioned, except for facial pain in men (Sipila et al, 2001).

Anger style and alexithymia may influence an individual's ability to cope with pain as a stressor.

9.II. THE STUDIES

The following chapters report three sequential studies, introduced below.

9.II.i. **The First Study**

The first study (**Chapter 10**) compared a *chronic* pain condition, temporomandibular disorder, with an *acute* pain condition, post-extraction pain, on measures of pain, disability and distress. TMD patients have been reported as more likely to suffer from anxiety and depressive disorders (**Chapter 2**), whilst pre-operative anxiety has been shown to predict post-operative pain in third molar surgery patients (**Chapter 3**). In addition, *subjective* self-reported disability in the TMD group was compared with *objective* clinical signs, since the usefulness of these signs has been called into question (**Chapter 2**).

The study also examined the relations between pain-related *cognitions*, disability and mood in TMD patients of varying chronicity. This research was intended to identify factors affecting mood and disability as the basis for a more appropriate intervention than those currently available.

9.II.ii. **The Second Study**

Having made a comparison with an acute condition of known cause and predictable course, an attempt was now made to assess the contribution of beliefs about the cause and course of TMD to the report of associated pain. A second comparison group, of primary headache sufferers, would illustrate the influence of pain location, i.e. jaw or head. The second study (**Chapter 11**) sought to determine the relations between pain intensity and character, and illness perceptions (cause, consequence, time-line and cure), and to compare these variables across two groups: TMD and Headache patients.

A longitudinal investigation (**Chapter 12**) was then conducted into the influence of cognitions on *change* in pain, disability and distress. In addition, putative mediating factors were assessed, including coping style, anger style and alexithymia.

9.II.iii. **The Third Study**

An attempt was then made to draw together the findings of the literature reviewed above and the results of the questionnaire-based studies into a psycho-education programme for TMD patients. Specific disability factors, correlates of distress, negative perceptions, and maladaptive coping strategies, were addressed in a programme focused on enhancement of self-efficacy and perceived control over pain, and reduction of anxiety (c.f. management strategies for acute dental pain, **Chapter 3**).

The aim of this project (**Chapter 13**) was to develop and pilot a simple, cost-effective, evidence-based management programme for TMD, using CD-ROM. A comparison group received adjunctive relaxation training, known to be effective in the management of this disorder. This approach was based on the transtheoretical model of behaviour change, developed from studies of smoking cessation (Prochaska & DiClemente, 1983) which proposes four stages of behaviour change: *precontemplation*, *contemplation*, *action*, and *maintenance*.

9.III. STUDY GROUPS

9.III.i. TMD Patients

TMD patients were recruited from Oral Surgery and Oral Medicine departments in several London hospitals and from one specialist clinic, after ethical approval and informed consent (**Appendix I**). Initially it had been intended to recruit TMD patients directly from General Dental Practitioners but the response was so poor as to make this impossible. However, the second study sample and the majority (90%) of the first study sample were from secondary referral centres for oral complaints (i.e. only 10% from the Facial Pain Clinic), and were interviewed prior to seeing a specialist, rather than from tertiary referral centres which often receive long-term patients with intractable problems (von Korff & Simon, 1996).

TMD patients were examined, according to the research diagnostic criteria for temporomandibular pain/dysfunction (**Appendix IV**: Dworkin & LeResche, 1992). Included were mandibular deviation on opening; joint sounds on opening, closing, lateral and protrusive excursions; tenderness to palpation of the joint and associated musculature; unassisted and assisted maximal jaw opening, and overbite (the vertical overlap of the lower central incisors by the upper central incisors). To assess jaw opening, the vertical distance between the tips of the upper and lower central incisors is measured both on asking the patient to open as wide as they can and then on gently attempting to manually increase that opening. The latter is considered to be a relatively objective measure since some degree of the 'guarding', that many patients exhibit, is overcome. Guarding is the unwillingness to open the jaw too wide in case it should hurt, and is therefore subjective. It will be evident, however, that even the assisted measure is subject to the patient's reaction and cooperation.

9.III.ii. **Post-Extraction Patients**

In the first study, 40 third molar patients were recruited from the Eastman Dental Hospital and University College Hospital Oral & Maxillofacial Surgery departments. Patients were approached prior to surgery and invited to complete the questionnaire three days post-extraction. Wisdom tooth patients generally report some degree of discomfort, maximally at 48 hours post-surgery.

9.III.iii. **Headache Patients**

Fifty-one headache patients were recruited from the Neurology departments of Wexham Park Hospital, Slough, Berkshire; and the Royal Berkshire Hospital, Reading, Berkshire. Subjects satisfied the ICD-10 diagnostic criteria for migraine or tension-type headache (**Appendix V**, IHS, 1997). Although it is accepted that these conditions differ in terms of pain character and frequency, both are idiopathic, of unknown duration and their prevention and management are incomplete. A mixed group therefore satisfied the purposes of the study.

Patients outside the age range of 18 to 70 years, or with a history of other chronic or psychiatric illness, were excluded.

9.IV. **MEASURES**

9.IV.i. **Age, Gender and Demographic Characteristics** were recorded, including marital status, occupation and years in full-time education.

Education level was included to assess any effect on pain-related beliefs.

9.IV.ii. **Pain Duration** (months since onset) **and Frequency** (days in the preceding month with pain).

These factors are important in establishing a diagnosis of chronic pain.

9.IV.iii. **Oral Health Impact Profile (OHIP,** see p.31**)**

In the first study, disability was measured with a 46-item Oral Health Impact Profile (OHIP), which has been developed to assess the social impact of oral disorders. Subscales include functional limitation, pain, psychological discomfort, physical disability, psychological disability, social disability and handicap. Its measurement properties have been established in older age groups and modification is needed for younger dentate subjects, by omission of denture-related items.

9.IV.iv. **Disability Checklist**

In the second study, disability was measured with a symptom checklist, derived from OHIP factors endorsed by TMD patients in the first study, together with defining characteristics for headache diagnosis (IHS, 1997).

9.IV.v. **McGill Pain Questionnaire (SF-MPQ)**

Current pain intensity was recorded by means of the short-form McGill Pain Questionnaire (SF-MPQ, Melzack, 1987). This is a valid and reliable instrument for outpatient groups, incorporating both a visual analogue scale (VAS) and a verbal rating scale (VRS) (Wilkie et al, 1990; Turp et al, 1997). The McGill Pain Questionnaire has been widely used as an assessor of both quality and intensity of

pain, and both its long and short forms are considered valid and reliable, with the short-form avoiding the more education-sensitive and culture-specific items found in the original (Pincus & Madland, 2000). Pain quality is considered in two dimensions: sensory and affective, yet few attempts have been made to confirm this structure in the short-form version.

9.IV.vi. **Hospital Anxiety and Depression scale (HAD,** see p.117**)**

The 14-item Hospital Anxiety and Depression scale (HAD, Zigmond & Snaith, 1983) assessed mood. A valid and reliable measure of both 'anxious' and 'depressive' symptoms in outpatient populations, the HAD is considered to be free of potentially confounding somatic items. Patients recording anxiety or depression scores of 7 or less are considered to be non-cases, scores of 8 to 10 are doubtful psychiatric cases and scores of 11 or more definite psychiatric cases. Although such scales do not equate to a psychiatric diagnosis, so-called 'false-positives', ie. 'cases' according to self-report scales but not to psychiatric interview, have been shown to display greater psychopathology than true-negatives whilst not differing from true-positives on measures of psychosocial dysfunction (Gotlib et al, 1995).

9.IV.vii. **State-Trait Anxiety Inventory (STAI-6)**

In the third study, anxiety was measured using this more widely used scale for better comparison with other studies. Short-form scores are similar to the full 20-item scale, with acceptable reliability, and sensitivity to different degrees of anxiety (Marteau & Bekker, 1992).

9.IV.viii. **Beck Depression Inventory (BDI,** see p.116)

This valid and reliable scale is the most widely used depression scale, and was substituted for the HAD in the third study, agaian for comparison with other studies, and sensitivity to change.

9.IV.ix. **Pain Beliefs Questionnaire (PBQ,** see p.100)

Patients' general beliefs about pain were assessed in the first study using the 12-item Pain Beliefs Questionnaire (PBQ, Edwards et al, 1992). This scale has been designed to measure beliefs about the aetiology and prognosis of pain symptoms, and validated in chronic pain groups. A distinction is made between 'organic' beliefs, where pain is attributed to an internal cause beyond the individual's control, and 'psychological' beliefs, where pain is attributed to factors affected by the individual and his/her environment within their control. Such a distinction might be drawn between the two study groups, involving an acute pain condition of known cause and an idiopathic chronic condition, and, hence, this questionnaire seems eminently suited for the first study.

The PBQ subscales have also been found to correlate with locus of control factors: "organic" with "chance" and "powerful others"; and "psychological" with "internal" locus of control (MHLC, Edwards et al, 1992).

9.IV.x. **Illness Perceptions Questionnaire (IPQ,** see p.101)

The Illness Perceptions Questionnaire (IPQ: Weinman et al, 1996) was developed to assess patients' representations of illness in order to improve the understanding of illness-related coping and to develop interventions to facilitate self-management in

chronic illness. The IPQ is a theoretically derived measure comprising five subscales: *identity* - the symptoms the patient associates with the illness; *cause* - personal ideas about aetiology; *consequences* - expected effects and outcome; and *control / cure* - how one controls or recovers from the illness.

The IPQ might thus provide an interesting comparison between two chronic idiopathic conditions: TMD and headache. The IPQ has not previously been used in these groups but has shown illness perceptions to be more explanatory of variance in disability and distress in Chronic Fatigue Syndrome than are coping strategies (COPE, Moss-Morris et al, 1996)

9.IV.xi. **Self-Efficacy Scale (SES)**

This is a brief scale for general perceived self-efficacy (Jerusalem & Schwarzer, 1992), which has been previously used in conjunction with interventions designed to enhance self-efficacy, and appears to be sensitive to change.

9.IV.xii. **Multidimensional Health Locus of Control scale (MHLC,** see p.98**)**

A valid and reliable measure of this belief construct (Wallston et al, 1978).

9.IV.xiii. **Coping Strategies Questionnaire (CSQ,** see p.86**)**

Patients' endorsement of pain-coping strategies and pain control was measured using the 44-item Coping Strategies Questionnaire (Rosenstiel & Keefe, 1983), which has been used in a number of investigations (for example: Nicassio et al, 1995; ter Kuile et al, 1995a,b; Snow-Turek et al, 1996). The CSQ has the advantage of being pain-specific, i.e. in specifying to the subject that their pain is the stressor for which they

are reuiqed to endorse coping strategies. Whilst the Vanderbilt Pain Management Inventory is similarly pain-specific, its "active" and "passive" subscales might be construed as over simplistic. The CSQ's "catsatrophising" factor, though controversial as a coping strategy, has widely replicated associations with psychological distress (for example: Dozois et al, 1995), and with pain report (Hill et al, 1995). 'Active' strategies of the CSQ ("coping self-statements", "reinterpreting sensations") have shown positive associations with perceived control over pain (SOPA, Haythornthwaite et al, 1998). In addition, different strategies have been demonstrated in pain groups of different chronicity (Main et al, 1996), suggesting changes over time; and changes in coping have been seen with cognitive therapy (ter Kuile et al, 1995; Turner et al, 1995), making the scale ideally suited to the later studies too.

9.IV.xiv. **Perceived Stress Scale (PSS-10)**

This is a brief and widely used measure of general perceived stress (Cohen et al, 1983). A general scale is desirable so as to avoid issues of cause and effect with pain symptoms, by not specifying a stressor.

9.IV.xv. **State-Trait Anger Expression Inventory (STAXI)**

This scale is the most widely used of its type and has a three factor solution comprising: anger expression, anger repression, and anger control (Spielberger, 1988).

9.IV.xvi. **Toronto Alexithymia Scale (TAS-20)**

This is a revised and shortened version of the original scale which demonstrated good internal consistency and test-retest reliability. The three-factor structure has been demonstrated in clinical and non-clinical populations using confirmatory factor analysis (Bagby et al, 1994).

9.IV.xvii. **Pain Stages of Change Questionnaire (PSOCQ)**

Designed to assess an individual's readiness to adopt a self-management approach to their chronic pain condition (Kerns et al, 1997), this scale is based on the transtheoretical model of behaviour change, with a 4-factor solution approximating to the four stages of the model: precontemplation, contemplation, action, and maintenance (Prochaska & DiClemente, 1983). The authors found the scale to be internally consistent and stable over time, and there was also support for each factor's discriminant and criterion-related validity. The clinical usefulness of the PSOCQ has yet to be established, however. In a sample of 110 diverse chronic pain patients and 119 fibromyalgia patients, individuals classified as being in the contemplation, action or maintenance stages according to the PSOCQ did not differ significantly on measures of beliefs and coping (SOPA, CPCI; Jensen et al, 2000). However, cluster analysis of a group of 177 arthritis patients identified five distinct subgroups, generally consistent with what might be expected, based on the transtheoretical model of change (Keefe et al, 2000b).

9.V. DATA ANALYSIS

Data were analysed by means of the Statistical Package for the Social Sciences (SPSS). To determine the structural characteristics of the questionnaires in these samples and as a data reduction exercise, principal component analyses were carried out, involving Varimax rotation; factors with Eigenvalues greater than 1.0, and items loading above 0.5 on one factor only, were included. Internal reliability was calculated for each factor using Cronbach's alpha, where an alpha greater than 0.65 was considered to reflect adequate internal consistency.

Correlations were performed using the Pearson correlation coefficient. Hierarchical Multiple Regressions and Logistic Regressions were carried out to predict the continuous and categorical variance respectively. Entry into the regression was based on significant correlation ($p=<0.01$) with the dependent variable. Order of entry was determined by the hypothetical schemata (**Figures 9.I,II,III.**). Stepwise Multiple Regression Analyses were also performed, in order to derive purely statistical comparisons.

9.VI. HYPOTHESES

1. In the first study, it was hypothesized that, in the TMD group, the clinical signs of joint sounds and jaw mobility would bear little relation to self-reported pain and disability (MPQ, OHIP), in accordance with the wide prevalence of these features in healthy populations (Pollmann, 1993; Szentpetery, 1993). Pains on palpation of joint and muscles were expected to relate more closely (i.e. positively correlate) to the questionnaire scores, being highly subjective responses evoking the prime symptoms of TMD.

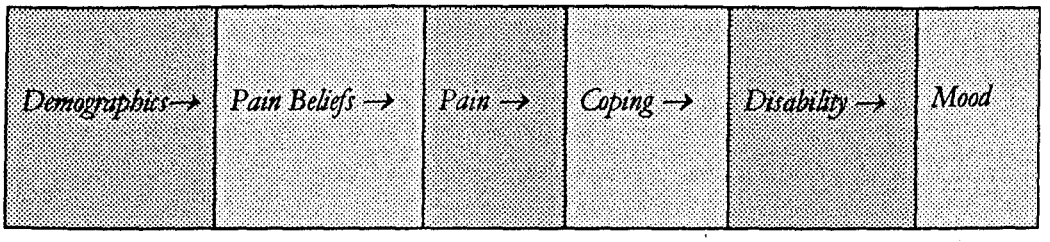
OHIP scores for this TMD group were expected to replicate those of the previous small Canadian group (Murray et al, 1996).

2. It was hypothesized that TMD and post-extraction patients would differ in their report of disability on the OHIP. The TMD group was expected to report greater psychological discomfort, psychological disability, social disability and handicap; whilst the post-extraction group would report greater functional limitation, pain, and physical disability, thus reflecting differences between a psychosocially disabling chronic condition and a physically disabling acute condition. In addition, the post-extraction group was expected to report greater pain intensity on the MPQ, as might be anticipated for a single episode rather than a recurrent experience.

3. In assessing their influence on pain, disability and distress in TMD, factors were hypothesized to act sequentially in a linear model. Although an oversimplification, in that bi-directional, reciprocal and cyclical influences may also

be involved, a linear model determined by theoretical concepts includes the most salient contributory factors and their relations. A hypothetical schema for the relations between the factors considered is illustrated in **Figure 9.I.**

Figure 9.I. A schema to illustrate the hypothesised relations between variables:



The order is derived thus: pre-existing, demographic factors are considered first, followed by beliefs about the cause of the pain, which, via appraisals such as locus of control and self-efficacy, influence the reported intensity of pain. Pain then prompts the adoption of specific coping strategies to, in turn, affect perceived disability and ultimately mood.

Hierarchical multiple regression analysis will establish the significance of each factor and relationship in contributing to distress.

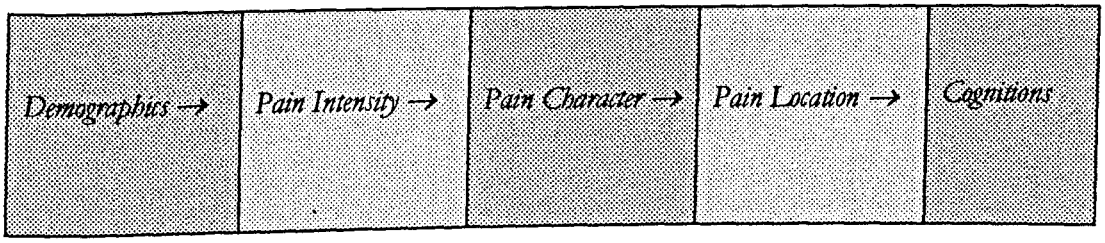
4. In the second study, it was hypothesized that causal attributions would categorise into psychological and organic components, and that the headache group would preferentially endorse the former, and the TMD group the latter, in line with the widely held belief in "tension headache" but lack of such a popular association in TMD.

5. It was hypothesized that principal component analysis of both groups' scores on the MPQ pain descriptors would derive a similar three-factor structure to that intended in the scale, i.e. intensity, sensory, and affective.

6. The intensity subscale of the MPQ was expected to correlate with greater organic pain beliefs and lesser psychological pain beliefs for both groups, from the theory that the experience of intense pain would provoke beliefs in an underlying pathology, similar to the greater intensity of pain in an acute condition of known cause.

7. The development of pain cognitions was hypothesized in a linear model. **Figure 9.II.** illustrates the putative schema. Pain intensity is followed in order of hypothesized importance by pain character, which is followed by pain location; so that the influence of intensity, on beliefs about cause, consequence, and time-line, is moderated by character and location, namely face or head.

Figure 9.II. Schema for hypothesised relations between variables influencing cognitions:



8. TMD and headache groups were expected to show modest and comparable improvement in the primary outcome measures of pain, disbility and distress, over six months, in line with other studies (Turner et al, 1995; ter Kuile et al, 1995).

9. Improvements in pain, disability and distress were expected to be associated with reductions in perceived consequence and time-line (i.e. persistence), increased endorsement of active coping strategies, and reductions in passive coping strategies.

10. Improvement over six months was expected to be predicted by belief in psychological cause of pain, and persistence of symptoms predicted by organic causal belief in the absence of any findings of pathology.

11. Alexithymia and repressive anger style were expected to negatively influence improvement in primary outcome, in both groups.

12. **Figure 9.III.** shows a putative schema, to be demonstrated in hierarchical multiple regression analyses for the primary outcomes.

Figure 9.III. A schema to illustrate the hypothesised relations between variables:

Experience→	*Stressors→*	*Assessment→*	*Coping→*	*Reaction*
Demographics	Past pain Stress	Illness perceptions	Alexithymia Anger style Coping strategies	Current pain Disability Mood

13. In the third study, it was hypothesized that the two experimental conditions would be equally effective in reducing pain, disability and distress, and both more effective than the attention placebo condition. Primary outcome improvements were expected to be associated with modest ameliorations in pain-related cognitions, including locus-of-control and self-efficacy.

Chapter 10. DISABILITY AND DISTRESS IN TMD IN COMPARISON WITH POST-EXTRACTION PATIENTS

10.I. AIMS

The first aim was to investigate the relationships between self-reported disability, as measured by the Oral Health Impact Profile (OHIP), clinical assessments, and self-reported symptoms in TMD patients, including the intensity scales of the McGill Pain Questionnaire (MPQ), and using a systematic approach to classifying the symptoms of TMD (RDC). In addition, reliability and validity of the OHIP were considered in comparing responses in this group of TMD patients with those of a small previous group (Murray et al, 1996), and with those of an acute orofacial pain group respectively.

The study also investigated the relationships between pain beliefs, coping strategies, disability and mood in TMD patients of varying chronicity.

10.II. PARTICIPANTS

10.II.i. TMD Patients

Eighty newly-referred TMD patients (79% females) were recruited from the Oral & Maxillofacial Surgery departments of: the Eastman Dental Hospital, London; University College Hospital, London; the Royal Free Hospital, London; and the Whittington Hospital, London; as well as from the Oral Medicine department and Facial Pain Clinic (10%) of the Eastman Dental Hospital.

TMD patients were examined, according to the research diagnostic criteria for temporomandibular pain/dysfunction (**Appendix IV**: Dworkin & LeResche, 1992).

10.II.ii. **Post-Extraction Patients**

In addition, 40 third molar patients were recruited from the Oral & Maxillofacial Surgery departments of: the Eastman Dental Hospital, London; and University College Hospital, London. Patients were approached prior to surgery and invited to complete the questionnaire three days post-extraction. As there were no routine post-operative review arrangements for the post-extraction patients, questionnaires from this group were returned by post in stamped addressed envelopes provided. Approximately 50% only of questionnaires were returned. The high drop-out reflects the acute nature of the discomfort involved and perceived lack of relevance of the questionnaires to individuals who so quickly recover. Ethical permission to pursue patients and encourage compliance was not sought, and the drop-out had to be accepted.

Information letter, consent form and questionnaire are included in **Appendix I.**

10.III. MEASURES

Age, Gender and Demographic Characteristics including marital status, occupation and years in full-time education.

Oral Health Impact Profile (OHIP: 46 items scored 0 = "never" to 4 = "very often"; pp.30, 136)

Pain Beliefs Questionnaire (PBQ: 12 items scored 0 = "never" to 5 = "always"; pp.100, 138)

Coping Strategies Questionnaire (CSQ: 44 itmes scored 0 = "not at all" to 6 = "very often / completely"; pp.86, 140)

McGill Pain Questionnaire (SF-MPQ: 15 descriptive adjectives scored 0 = "none" to 3 = "severe"; visual analogue scale 0 = "no pain" to 100 = "worst possible pain"; present pain intensity verbal rating 0 = "no pain" to 5 = "excruciating"; p.137)

Hospital Anxiety and Depression scale (HAD: 14 items – 7 anxious and 7 depressive – 0 e.g. "not at all" to 3 = "very much indeed", pp.117, 137)

Analgesic Use (TMD patients only, scored 0 = "no" or 1 = "yes").

Clinical Signs (TMD patients only; based on RDC/TMD; see **Appendix IV**).

10.IV. RESULTS

10.IV.i. Group Means and Percentages

Table 10.I. shows mean scores for both groups on demographic factors. There was a significant difference in age between the TMD and post-extraction groups (means 38 and 29 years respectively, $p<0.001$). **Table 10.III.** shows mean scores for both groups on OHIP factors and MPQ pain intensity scales. The post-extraction group scored significantly higher than the TMD group on the Functional Limitation (15.5, SD 7.4 vs. 9.6, SD 6.1,) and Physical Disability scales (13.5, SD 9.5 vs. 8.0, SD 6.2,).

Table 10.IV. shows scores on symptom duration and jaw opening for the TMD group. Jaw opening measures include overbite. Fifty-nine per cent of TMD patients exhibited palpable jaw sounds, 56% reported pain on palpation of the temporomandibular joint, 48% reported pain on palpation of the masticatory muscles, and 26% had no pain on palpation at the time of examination, despite a history of such.

Table 10.I.
Demographic characteristics for both groups: percentages, means (and SDs):

	TMD group	**Post-Extraction group**	Difference
sex	79% female	63% female	NS.
age (years)	37.6 (13.3)	28.6 (8.6)	$p<0.001$
years in full-time **education**	14.2 (4.1)	15.3 (3.6)	NS.

Table 10.II. Pain and mood scores for the TMD group (mean, SD and range):

	mean	SD	minimum	maximum	n*
duration (months)	29.2	37.2	1	180	80
current pain (visual analogue)	32	26	0	100	78
current pain (verbal rating)	1.5	1.1	0	5	79
anxiety	8.4	4.7	0	21	80
depression	4.8	3.9	0	18	80

* NB. the number of subjects varies because some questionnaires were incomplete.

Table 10.III.
Oral Health Impact Profile and pain scores for both groups: means (and SD's):

	TMD group	**Post-extraction group**	Difference
N	80	40	
OHIP Subscales: Pain	17.3 (6.7)	19.6 (7.0)	NS.
Functional limitation	9.6 (6.1)	15.3 (7.4)	$p<0.001$
Psychologic discomfort	8.9 (5.4)	7.6 (5.8)	NS.
Physical disability	8.0 (6.2)	13.5 (9.5)	$p<0.001$
Psychologic disability	11.1 (6.1)	9.7 (6.5)	NS.
Social disability	5.4 (4.8)	7.0 (5.1)	NS.
Handicap	5.3 (5.8)	7.2 (5.7)	NS.
MPQ Pain Scores: VAS (0-100)	32 (26)	31 (22)	NS.
VRI (0-5)	1.5 (1.1)	2.0 (1.1)	$p<0.05$

Table 10.IV.
Symptom duration and jaw opening scores for the TMD group (mean, SD and range):

	mean	SD	range
duration (months)	29.2	37.2	1 to 180
jaw opening, unassisted + overbite (mm)	38.6	8.7	17 to 52
jaw opening, assisted + overbite (mm)	39.6	8.3	18 to 52

10.IV.ii. **Relationship between the disability factors, as measured by the OHIP, pain and clinical signs**

There were no significant relationships between OHIP subscale scores and clinical signs. All OHIP subscales except for 'functional limitation' and 'physical disability' correlated significantly with both the visual analogue and verbal rating scales of pain intensity, as measured by the McGill (**Table 10.V.**). There was also a significant difference in means for both pain measures between those TMD subjects who reported pain on palpation of the TMJ and those who didn't (VAS: 41, SD 25 vs. 21, SD 25; VRI: 1.8, SD 1.1 vs. 1.1, SD 1.0). There were no such differences for the presence or absence of muscle pain or joint sounds on palpation.

Table 10.V. Correlation between the OHIP factors and pain scores on the McGill Questionnaire in the TMD group ($p<0.01$):

	MPQ: VAS	MPQ: VRI
Pain	.42	.41
Psychological discomfort	.51	.44
Psychological disability	.51	.45
Social disability	.43	.41
Handicap	.35	.38

10.IV.iii. **Comparison with previous samples**

Mean OHIP scores were also calculated using the same method as previous researchers. This involves grouping the responses of 'fairly often' and 'very often' to items, and grouping the three other possible responses. In this way the responses to the OHIP items are made into a binaryscore. It should be noted that this scoring sytem is somewhat crude as it reduces the information available from the 5-point Likert-type scales to a binary measure. **Table 10.VI.** compares the means for the TMD and post extraction groups with those of the two pain groups from the previous study. It is of note that the means for the TMD groups are almost identical. The scores for the seven subscales were also calculated and although not subjected to any statistical analysis, the TMD group in the current study showed greater functional limitation, physical disability, psychological disability, social disability and handicap.

10.IV.iv. **TMD Group Only: Means**

Tables 10.I. & II. show mean scores for the TMD group on demographic variables (mean age 38 years, SD 13; 14 years in full-time education, SD 4), symptom duration (29 months, SD 37), pain intensity (VAS 32mm, SD 26; VRS 1.5 = 'mild'/'discomforting', SD 1.1) and mood. The mean scores for anxiety and depression, as measured by the HAD, were 8.4 (SD 4.7) and 4.8 (SD 3.9) respectively.

10.IV.v. **Pain Beliefs**

The Pain Beliefs Questionnaire was subjected to a principal component analysis, which yielded a 4-factor solution. Two of the factors did not demonstrate satisfactory levels of internal reliability (Cronbach's alpha .39 and .32). Of the two reliable factors, one factor (Cronbach's alpha = .75) consisted of three items all reflecting psychological beliefs ('being anxious makes pain seem worse', 'when relaxed, pain is easier to cope with' and 'feeling depressed makes pain seem worse'). The second factor (Cronbach's alpha = .67) consisted of three items reflecting organic beliefs ('experiencing pain is a sign that something is wrong with the body', 'being in pain prevents you from enjoying hobbies and social activities' and 'pain is a sign of illness').

Table 10.VI. Comparison between the TMD and post extraction group and two previous study groups on the Oral Health Impact Profile, using a binary scoring system.

Total scale: mean number (and SD) of 'very often' / 'fairly often' responses.

Subscales: percent of subjects with one or more 'very often' / 'fairly often' responses:

	Present TMD group	Previous TMD group	Other facial pain group	Post-extraction group
N	80	c.30	c.30	40
Total scale	7.3 (6.6)	7.2 (8.5)	9.5 (6.9)	8.5 (7.7)
Subscales:				
Pain	90.4	-	-	84.2
Functional limitation	61.8	54.8	33.3	65.0
Psychologic discomfort	57.7	58.1	79.2	48.7
Physical disability	68.4	61.3	54.2	70.3
Psychologic disability	65.8	51.6	75.0	59.0
Social Disability	34.7	29.0	55.0	52.6
Handicap	30.3	25.8	58.3	44.7

NB. 'Functional limitation' and 'social disability' subscales in previous study included one extra item each not included in present study.

10.IV.vi. **Coping Strategies**

The principal component analysis of the CSQ yielded a 9-factor solution accounting for 70% of the variance, Eigenvalues greater than 1.0. In common with the original Factor Analysis by the authors of the scale, the first factor, which accounted for 19.0% of the variance (Eigenvalue of 8.4, Cronbach's alpha .91) consisted of items chiefly reflecting Catastrophising (for example: 'it's terrible and it's never going to get better', 'I feel like I can't go on'). A second factor corresponded to 'reinterpreting pain sensations' (seven items, 17.9% of the variance, Eigenvalue 7.9, Cronbach's alpha .87; for example: 'I try to feel distant from the pain, almost as if it was in someone else's body'). A third factor approximated to 'ignoring pain sensations' (six items, 7.6% of the variance, Eigenvalue 3.4, Cronbach's alpha .85; for example: 'I don't pay any attention to the pain'); a fourth to 'increased behavioural activities' (four items, 5.4% of variance, Eigenvalue 2.4, Cronbach's alpha .75; 'I do something active, like household chores or projects'); a fifth 'praying' (two items, 4.8% of variance, Eigenvalue 2.1, Cronbach's alpha .85; 'I pray to God it won't last long'); a sixth 'diverting attention' (three items, 4.2% of variance, Eigenvalue 1.9, Cronbach's alpha .87; 'I think of things I enjoy doing'); a seventh 'coping self-statements' (three items, 3.9% of variance, Eigenvalue 1.7, Cronbach's alpha .81; 'I tell myself that I can overcome the pain'); an eighth which proved unreliable (three items, 3.6% of variance, Eigenvalue 1.6, Cronbach's alpha .61); and a ninth which loaded the pain control questions (3.3% of variance, Eigenvalue 1.4, Cronbach's alpha .70; items 43. 'I feel I have control over my pain'and 44. 'I am able to decrease the pain').

10.IV.vii. **Disability**

A principal component analysis of the OHIP was also conducted, in order to assess the factor structure in the TMD group. Analysis yielded a 6-factor solution: one factor approximated to four of the authors' subscales (psychological discomfort, psychological disability, social disability, handicap). This factor consisted of 14 items (Cronbach's alpha .95). A second factor comprised five items chiefly concerning problems with eating (Cronbach's alpha .85); a third (five items, Cronbach's alpha = .85) concerning self-image; a fourth (three items, Cronbach's alpha .74) concerning oral pain; a fifth (three items, Cronbach's alpha .84) concerning problems with speech; and a sixth (three items, Cronbach's alpha .79) concerning taste and digestion. Apart from the first 'mood' factor and the 'oral pain' factor, which corresponded to the authors' pain factor, the factor structure in this group bore little resemblance to the authors' original structure.

10.IV.viii. **Correlation Analysis**

There were significant correlations (p=<.01) between anxiety and depression scores (HAD), pain, and factors derived from the principal component analyses for the PBQ, CSQ and OHIP. Anxiety and depression had a Pearson's correlation coefficient of .58. The two present pain intensity measures, the visual analogue scale and the verbal rating scale, correlated at .73. Anxiety correlated with pain (VAS, .34; VRS .37); psychological pain beliefs (PBQ1, .32); catastrophising (CSQ1, .50); mood impact (OHIP1, .69); self-image impact (OHIP3, .32); impact on speech (OHIP5, .34); and impact on taste (OHIP6, .46). Depression correlated with catastrophising (.60); praying (CSQ5, .33); mood impact (.51); impact on eating (OHIP2, .36); self-image impact (.45); and impact on taste (.60). It is of note that there were no significant correlations with any demographic variables.

10.IV.ix. **Factors Predictive of Anxiety and Anxious Mood**

Hierarchical Multiple Regression analysis was performed to assess the relative contributions of correlating factors in the TMD group to the variance in anxious mood and anxiety, accoreding to the putative schema (**Figure 10.I**). No demographic variables were found to correlate with anxious mood so these were not entered into the hierarchical Multiple Regression. The two measures of pain, entered on the first step, accounted for 14% of the variance of anxious mood (See **Table 10.VII**). The 'Psychological' factor of the PBQ entered on the next step accounted for a further 9% and Catastrophising from the CSQ accounted for an additional 14%. When the four disability factors from the OHIP were entered, two (Mood and Speech) were found to make significant contributions to the explanation of anxious

mood and together they accounted for an additional 12% of the variance. Depressed mood from the HAD was entered on the final step and it added a further 5%. The final equation accounted for 55% of the variance in anxious mood.

A Logistic Multiple Regression was also performed to examine which factors predicted clinical anxiety (Cut-off of 8 on the HAD; **Table 10.VIII.**). In this analysis pain alone predicted 73% of possible cases. The addition of psychological pain beliefs predicted a further 5%. It is of note that catastrophising and depressed mood, although significant predictors of non-cases and hence contributing to the overall prediction, added nothing to the prediction of cases. The disability factors (Self-image, Speech and Taste disturbance) increased the predictive power to 83%.

10.IV.x. **Factors Predictive of Depression and Depressed Mood**

In the absence of significant correlations, demographic variables, pain report and pain beliefs were omitted from the hierarchical Multiple Regression analysis for depressed mood (**Table 10.IX.**). On the first step, Catastrophising accounted for 34% of the variance. Disability factors, significantly Taste Disturbance, accounted for a further 16%, and anxious mood 3%. The final equation accounted for 54% of the variance.

In the logistic analysis for clinical depression (**Table 10.X**), Catastrophising predicted 39% of possible cases. The disability factors (significantly Taste Disturbance) increased this to 72%. Anxious mood had no effect on case prediction but a small effect on the prediction of non-cases.

Table 10.VII.

Hierarchical Multiple Regression analysis of variables on anxious mood, n=73:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	pain (visual)	.124	.463		
	pain (verbal)	.309	.070	.145	.145
2	PBQ1 (psychological)	.317	.003	.235	.090
3	CSQ1(catastro -phising)	.427	.000	.377	.142
4	OHIP1 (mood)	.334	.019	.496	.119
	OHIP3 (self-image)	-.176	.122		
	OHIP5 (speech)	.218	.018		
	OHIP6 (taste)	.177	.103		
5	depression (HAD)	.333	.005	.548	.052

Final regression equation (F 10.71, df 9,63; p=.000)

Table 10.VIII.
Hierarchical logistic regression analysis of variables on clinical anxiety, n =73 (41 cases, 32 non-cases; HAD scores greater than 7 denote possible 'caseness'):

step	variable	sig.	exp (B)	classification table 0 = non-cases 1 = possible cases	% correct overall prediction
1	pain (visual)	.060	1.033	predicted 0 1	
	pain (verbal)	.540	1.266	observed 0 19 13 1 11 30	67.1
2	PBQ1 (psycho-logical)	.035	1.207	predicted 0 1 observed 0 20 12 1 9 32	71.2
3	CSQ1 (catastro-phising)	.003	1.125	predicted 0 1 observed 0 28 4 1 12 29	78.1
4	OHIP1 (mood)	.106	1.068		
	OHIP3 (self-image)	.000	.909	predicted 0 1	
	OHIP5 (speech)	.029	1.236	observed 0 27 5 1 7 34	
	OHIP6 (taste)	.000	.972		83.6
5	depression (HAD)	.020	1.553	predicted 0 1 observed 0 26 6 1 7 34	82.2

Table 10.IX.

Hierarchical Multiple Regression analysis of variables on depressive mood, n=76:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	CSQ1 (catastro-phising)	.625	.000		
	CSQ5 (praying)	-.045	.708	.340	.340
2	OHIP1 (mood)	.271	.828		
	OHIP2 (eating)	-.013	.892		
	OHIP3 (self-image)	.010	.921		
	OHIP6 (taste)	.446	.000	.501	.161
3	anxiety (HAD)	.267	.017	.535	.034

Final regression equation (F 13.34, df 7,68, p=.000)

Table 10.X.
Hierarchical logistic regression analysis of variables on clinical depression, n=76 (18 cases, 58 non-cases; HAD scores greater than 7 denote possible 'caseness'):

step	variable	sig.	exp (B)	classification table 0 = non-cases 1 = possible cases	% correct overall prediction
1	CSQ1 (catastro-phising)	.005	1.094		
	CSQ5 (praying)	.910	1.010	predicted 0 1 observed 0 54 | 4 1 11 | 7	80.3
2	OHIP1 (mood)	.222	.950		
	OHIP2 (eating)	.909	1.008		
	OHIP3 (self-image)	.067	1.163	predicted 0 1 observed 0 54 | 4 1 5 | 13	
	OHIP6 (taste)	.012	1.411		88.2
3	anxiety (HAD)	.036	1.247	predicted 0 1 observed 0 56 | 2 1 6 | 12	89.5

10.V. DISCUSSION

The stated hypotheses were:

1. In the TMD group, the clinical signs of joint sounds and jaw mobility would bear little relation to self-reported pain and disability (MPQ, OHIP), in accordance with the wide prevalence of these features in healthy populations (Pollmann, 1993; Szentpetery, 1993). Pains on palpation of joint and muscles were expected to relate more closely (i.e. positively correlate) to the questionnaire scores, being highly subjective responses evoking the prime symptoms of TMD.

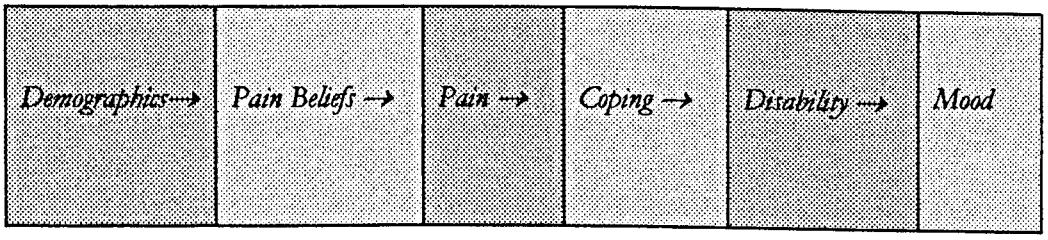
OHIP scores for this TMD group were expected to replicate those of the previous small Canadian group (Murray et al, 1996).

2. It was hypothesized that TMD and post-extraction patients would differ in their report of disability on the OHIP. The TMD group was expected to report greater psychological discomfort, psychological disability, social disability and handicap; whilst the post-extraction group would report greater functional limitation, pain, and physical disability, thus reflecting differences between a psychosocially disabling chronic condition and a physically disabling acute condition. In addition, the post-extraction group was expected to report greater pain intensity on the MPQ, as might be anticipated for a single episode rather than a recurrent experience.

3. In assessing their influence on pain, disability and distress in TMD, factors were hypothesized to act sequentially in a linear model. Although an oversimplification, in that bi-directional, reciprocal and cyclical influences may also be involved, a linear model determined by theoretical concepts includes the most

salient contributory factors and their relations. A hypothetical schema for the relations between the factors considered is illustrated in **Figure 9.I.**

Figure 9.I. A schema to illustrate the hypothesised relations between variables:



10.V.i. **OHIP Factors and Clinical Signs**

There were no significant relationships between OHIP factors and clinical signs. This is in line with previous findings that the course of TMD is unrelated to changes in clinical signs (Ohrbach & Dworkin, 1998). All OHIP factors correlated significantly with pain intensity, except for the functional and physical subscales, suggesting again that it is the psychosocial aspects of disability which relate to pain report, rather than the physical aspects. Arthralgia on palpation was related to the report of pain on the MPQ but myalgia and joint sounds were not. This may reflect a distinction between arthrogenous and myogenous disorder, with the latter being less clearly related to the report of pain *per se*, and with joint sounds being irrelevant.

Thus the first set of hypotheses was largely supported.

10.V.ii. **Group Differences on the OHIP**

Post-extraction patients reported greater functional limitation and physical disability than did TMD patients, which reflects the difference between an acute and a chronic

condition, and demonstrates the ability of the OHIP to distinguish such. This distinction is missed on using the previous binary scoring system. However, caution must be exercised on interpreting the results from the post-extraction group because of the self-selecting nature of the sample due to the high drop-out. It is likely that those individuals motivated to complete and rertum the questionnaire were those suffering the most from their experience.

Comparison of scores on the OHIP shows greater disability in this British TMD group than in a previous Canadian group, although significance is unknown. This is perhaps particularly surprising considering the Canadian sample came from a specialist facial pain clinic whereas the majority (90%) of the present subjects came from secondary referral centres catering for more general oral needs. In addition, no information on chronicity is available from the previous study.

The second set of hypotheses was partly supported, although TMD subjects were not significantly more disabled than were post-extraction subjects, in terms of psychological and social factors. Drop-out, possibly of less disabled subjects, from the post-extraction group may have obliterated any distinction.

10.V.iii. **Gender and Distress**

The mean age and gender distribution of the sample are comparable with other studies of TMD epidemiology (Locker & Slade, 1988). The prevalence rates for possible clinical anxiety and depression in this sample of TMD patients, as suggested by the HAD, were 58% and 23% respectively. These rates may be compared with those in other TMD groups; for example, 30% anxiety disorders and 23% affective

disorders (DSM-III-R, Gatchel et al, 1996). The high prevalence of potential clinical anxiety may reflect the low cut-off score used with the HAD.

10.V.iv. **Pain Ratings**

The mean pain intensity ratings are comparable with previous findings: for example 39mm on a 100mm VAS in a group of 157 TMD clinic patients (Orhbach & Dworkin, 1998). One limitation of the pain ratings is the report of current pain only: since TMD is an intermittent condition, a number of patients reported no pain (11.5% on the VAS; 21.5% on the VRS). The incorporation of a measure of recent pain experience, for example over the preceding month, might well have shown a greater influence of pain over psychological distress.

10.V.v. **Pain Beliefs**

The structures of the pain beliefs and coping scales were similar to those found for other pain groups (Edwards et al, 1992; Rosenstiel & Keefe, 1983), suggesting that common beliefs are held and common coping strategies employed across pain groups, although their relative endorsement may vary. Unfortunately, the two reliable factors obtained for the Pain Beliefs Questionnaire included only 6 out of the 12 items. This indicates that the original PBQ may be of limited use in this group of patients. The scale items may also be too transparent and subjects' responses affected by social desirability.

10.V.vi. **Coping Strategies**

The most significant coping strategy endorsed in the Coping Strategies Questionnaire was Catastrophising. No 'active' strategies were predictive of distress. This is in common with previous studies using the CSQ (Hill et al, 1995; Dozois et al, 1996). There has been some debate over whether or not catastrophising should be considered a coping strategy. Jensen et al (1991) considered it to be purely an appraisal, whilst Sullivan and D'Eon (1990) conceptualised it as another index of psychological distress / depression. Examination of the items included in this factor show it to be certainly a measure of the individual's perceived inability to cope with their pain, rather than an endorsement of specific coping strategies. However, despite all the subscale items reflecting depression (Sullivan & D'Eon, 1990), catastrophising differs from other mood scales in being *reported* as directly consequent to the experience of pain.

10.V.vii. **Disability Factors**

The structure of the Oral Health Impact Profile differed markedly from that described by the authors (Slade & Spencer, 1994). The principal component analysis derived mood impact, problems with eating, self-image impact, oral pain, problems with speech and disturbance in taste and digestion. The structural difference from the original scale, which was developed from general populations, suggests that the disability of TMD is qualitatively quite different from that of more common oral health problems. In the only previous study to use the OHIP for facial pain patients (Murray et al, 1996), the population was of mixed diagnosis, preventing the identification of specific disability factors in TMD.

10.V.viii. **Factors Associated with Anxious Mood**

Factors were analysed both for a continuous variable (anxious mood) and a dichotomous variable (potential anxiety cases versus non-cases). Individuals with more pain were found to have higher levels of anxious mood. This finding is in common with other research that has established a clear relationship between the report of pain and anxious mood (for example: McCracken et al, 1993; Casten et al, 1995). Interestingly, and perhaps surprisingly, subjects with stronger beliefs in a psychological influence on their pain tended to have higher levels of anxious mood. Catastrophising as a coping style has been found in other studies to be related to psychological well-being but this association has primarily been with measures of depressed mood or clinical depression (Jensen et al, 1991). In this study catastrophising was found to be related to anxious mood. One might speculate that the catastrophising reaction to TMD increases desperation and uncertainty regarding the course of the problems, thus serving to increase anxious mood. No other study specifically relating catastrophising to anxiety was identified.

When the OHIP factors were included in the equation, OHIP mood and problems with speech added significantly to the prediction of variance. In many ways it is unsurprising that the mood factor is associated with anxious mood, as measured by the HAD, since both are assessing the same construct. When the data were subjected to a stepwise regression, it was OHIP mood which appeared first and accounted for 41% of the variance. Why speech problems should add to the prediction of anxious mood is less clear. It may be that perceived difficulties with speech provoke anxiety because of the frequency and importance of speech in social situations.

When levels of anxiety were categorised into potential psychiatric cases (scores greater than 7) and non-cases (scores of 7 or less), the impact of pain became more apparent. Pain has been found, in other studies, to influence the likelihood of a diagnosis of anxiety disorder (for example: Gatchel et al, 1996). Pain's prediction of non-cases of anxiety was less accurate, however. That is to say there was a number of false-positives. The inclusion of catastrophising into the equation removed many of these.

10.V.ix. **Factors Associated with Depressed Mood**

The association of passive coping with depressed mood is consistent with that found in other chronic pain groups (for example: Snow-Turek et al, 1996). Catastrophising was highly predictive of non-cases of depression but less predictive of cases. That is to say there were, as a result, few false-positives but relatively many false-negatives. The prediction of cases was significantly improved by the inclusion of OHIP taste disturbance.

The association of perceived disturbance in taste and digestion with depressed mood / depression appears to be a new finding in TMD patients and is intriguing since the condition has previously been associated with mechanical, rather than sensory, impairment of eating. It might be possible that taste sensation is confused with pain, or more likely that this reported impairment is a reflection of the loss of pleasure derived from eating.

Thus some support was derived for the hypothetical schema of factors influencing psychological distress, with specific aspects of disability contributing to the variance.

Chapter 11. TEMPOROMANDIBULAR DISORDER AND HEADACHE: ILLNESS PERCEPTIONS, PAIN CHARACTER AND INTENSITY

11.I. AIMS

The second study sought to determine the relations between pain intensity and character, and illness perceptions (cause, consequence, time-line and cure), and to compare these variables across two groups: TMD and Headache patients.

11.II. PARTICIPANTS

Fifty-one TMD and fifty-one headache patients completed the questionnaires. All participants were aged 18 to 70 years and English speakers. Those suffering from other painful chronic illness were excluded. Informed consent and ethical approval was obtained. TMD patients were recruited from new referrals to the Oral & Maxillofacial Surgery departments of: University College Hospital, London; the Royal Free Hospital, London; the Whittington Hospital, London; Chase Farm Hospital, Enfield, Middlesex; and the Royal Berkshire Hospital, Reading, Berkshire. Participants satisfied the research diagnostic criteria (RDC/TMD, Dworkin & LeResche, 1992). Headache subjects were recruited from the Neurology departments of Wexham Park Hospital, Slough, Berkshire, and the Royal Berkshire Hospital, Reading, Berkshire. Patients were invited to complete the questionnaire at the time of their initial outpatient appointment.

11.III. MEASURES

Age, Gender and Demographic Characteristics including marital status, occupation and years in full-time education.

Pain Duration (months) **and Frequency** (days with pain during preceding month)

Illness Perceptions Questionnaire (IPQ: 41 items scored 1 = "strongly disagree" to 5 = "strongly disagree"; pp. 101, 139)

McGill Pain Questionnaire (SF-MPQ: 15 descriptive adjectives scored 0 = "none" to 3 = "severe"; visual analogue scale 0 = "no pain" to 100 = "worst possible pain"; present pain intensity verbal rating 0 = "no pain" to 5 = "excruciating"; p. 137)

11.IV. RESULTS

11.IV.i. Demographics and Pain History

Table 11.I. shows the demographics and pain history for both groups. The headache group's symptoms had started prior to those of the TMD group (duration: 72 months vs. 30 months).

11.IV.ii. Attributions of Cause

The first ten items of the Illness Perceptions Questionnaire asked subjects to describe their level of agreement with various possible causes for their condition. A principal component analysis was carried out for responses from the entire group to the initial scale, as a data reduction exercise. **Table 11.II.** shows how these responses converged into three factors, which may be termed *'Psychological'*, *'Physiological'* and *'External'*.

Table 11.I. Group means (and SD's) on the demographic and pain history factors. Significant difference denoted: *p<.05.

	TMD	HEADACHE	range
age (years)	37 (15)	40 (13)	18 - 70
gender (% female)	76	67	
education (years)	14 (4)	13 (3)	6 - 20
duration (months)	30 (58)	72 (116)*	1 – 540
frequency (days per month)	24 (10)	19 (12)	0 - 30

Table 11.II. Principal Component Analysis of Causal Factors (IPQ 1-10) for the entire group. Eigenvalues > 1.0; Coefficients > 0.6 for the allotted factor and < 0.4 for any other.
Rotated Factor Matrix (VARIMAX converged in 4 iterations):

	FACTOR 1	FACTOR 2	FACTOR 3
Eigenvalue	3.48	1.89	1.33
Percentage of variance	34.8 %	18.9 %	13.3 %
1. 'Germ or virus'	-.269	**.821**	.047
2. 'Diet'	.185	**.815**	.091
3. 'Pollution'	.201	**.779**	.060
4. 'Hereditary'	.368	**.603**	-.015
5. 'Chance'	.107	.223	**-.790**
6. 'Stress'	**.896**	.041	-.072
7. 'My own behaviour'	**.802**	.154	.220
8. 'Other people'	.375	.175	**.682**
9. 'Poor dental care'	.155	.222	**.627**
10. 'My state of mind'	**.836**	.142	.253

FACTOR 1 *'Psychological'*	FACTOR 2 *'Physiological'*	FACTOR 3 *'External'*
6. 'Stress'	1. 'Germ or virus'	5. 'Chance' (negative)
7. 'My own behaviour'	2. 'Diet'	8. 'Other people'
10. 'My state of mind'	3. 'Pollution'	9. 'Poor dental care'
	4. 'Hereditary'	

11.IV.iii. **Characterisation of Pain**

Table 11.III. shows the percentage of subjects who reported at least mild pain fitting each descriptor, as well as the group percentages and mean scores ('none' = 0 to 'severe' = 3). Melzack's criterion for a descriptor to represent a patient group is 33% use (Melzack, 1987).

Hence 'stabbing', 'hot/burning', 'splitting', 'fearful', and 'punishing/cruel', were not relevant to the TMD group, whilst 'gnawing' was not relevant to the headache group. In this analysis of two groups, a cut-off prevalence of 40% was used to determine the relevance of a descriptor to both. Thus 'gnawing', 'hot/burning', 'fearful', and 'punishing/cruel' were excluded from further analysis.

The remaining eleven adjectival descriptors were then subjected to a Principal Component Analysis for the entire group, and converged into three factors (see **Table 11.IV.**), which may be termed: 'constant', 'intermittent' and 'muscular'.

Table 11.III. Percent use of pain descriptors and group mean (and SD) intensity score of each word. Significant differences in means denoted: *p<.05, **p<.01, ***p<.001.

	% use			mean (and SD)	
Word	**All**	**TMD**	**Headache**	**TMD**	**Headache**
Throbbing / Pulsating	71	53	88	.94 (1.1)	2.1 (1.0)***
Shooting	54	45	63	.84 (1.1)	1.3 (1.0)*
Stabbing	49	31	73	.61 (1.0)	1.5 (1.3)***
Sharp	61	51	73	1.0 (1.2)	1.7 (1.2)**
Cramping	45	55	35	.92 (.98)	.61 (.96)
Gnawing	37	47	26	.84 (1.0)	.51 (.96)
Hot / Burning	29	20	37	.38 (.81)	.73 (1.1)
Aching	86	90	82	2.0 (.89)	2.0 (1.1)
Heavy	62	47	78	.86 (1.0)	1.9 (1.2)***
Tender	70	78	61	1.5 (1.0)	1.2 (1.2)
Splitting	51	23	80	.45 (.92)	2.0 (1.2)***
Tiring / Exhausting	60	43	78	.88 (1.2)	1.9 (1.2)***
Sickening	44	23	65	.39 (.83)	1.3 (1.2)***
Fearful	29	22	37	.39 (.85)	.67 (.95)
Punishing / Cruel	26	18	36	.37 (.87)	.70 (1.1)

Table 11.IV. Principal Component Analysis of Relevant Pain Descriptors (MPQ) for the entire group. Eigenvalues > 1.0; Coefficients > 0.5 for the allotted factor and < 0.5 for any other; Varimax rotation.

Rotated Factor Matrix (VARIMAX converged in 8 iterations):

	FACTOR 1 *'Constant'*	FACTOR 2 *'Intermittent'*	FACTOR 3 *'Muscular'*
Eigenvalues	4.01	1.58	1.34
Percentage of variance	36.5 %	14.4 %	12.2 %
MPQ items:			
Throbbing / Pulsating	**.709**	.205	-.193
Shooting	.084	**.871**	.095
Stabbing	.404	**.760**	-.013
Sharp	.145	**.796**	.168
Cramping	.047	.073	**.688**
Aching	.372	-.082	**.715**
Heavy	**.709**	-.048	.387
Tender	-.067	.357	**.625**
Splitting	**.665**	.438	.041
Tiring / Exhausting	**.701**	.146	.275
Sickening	**.768**	.153	.085

FACTOR 1 *'Constant'*	FACTOR 2 *'Intermittent'*	FACTOR 3 *'Muscular'*
Throbbing / Pulsating	Shooting	Cramping
Heavy	Stabbing	Aching
Splitting	Sharp	Tender
Tiring / Exhausting		
Sickening		

11.IV.iv. **Inter-Group Differences**

Table 11.V. shows inter-group differences in means on MPQ and *IPQ* factors. The headache group's pain was more intense than that of the TMD group (VAS: 66 vs. 48; PPI 3.3 vs. 2.3), and, specifically, 'constant' and 'intermittent' pains were more intensely reported, whilst 'muscular' pain was not (8.9 vs. 3.5, 4.5 vs. 2.5, 3.9 vs. 4.4, respectively). The headache group also perceived their symptoms to be of greater consequence than did the TMD group (23 vs. 18).

Table 11.V. Group means (and SD's) on the MPQ and *IPQ* factors. Significant differences denoted: *p<.05, **p<.01, ***p<.001.

	TMD	HEADACHE	**range**
'Constant' pain	3.5 (3.7)	8.9 (3.7)***	0 - 15
'Intermittent' pain	2.5 (2.7)	4.5 (3.2)***	0 - 9
'Muscular' pain	4.4 (2.2)	3.9 (2.2)	0 - 9
Intensity (VAS)	48 (27)	66 (24)***	0 - 100
Intensity (PPI)	2.3 (1.1)	3.3 (1.1)***	0 - 5
Psychological cause	7.9 (3.2)	8.3 (2.8)	3 – 15
Physiological cause	7.6 (3.3)	9.0 (3.0)*	4 – 20
External cause	7.7 (2.3)	6.8 (2.4)	3 - 15
Timeline	9.2 (2.4)	9.8 (2.1)	3 - 15
Consequence	18.3 (5.1)	23.1 (4.8)***	7 - 35
Control / Cure	19.8 (2.7)*	18.4 (2.5)	6 - 30

11.IV.v. **Correlation of IPQ and MPQ Scores**

Correlation tests were performed across the entire sample (**Table 11.VI.**) for four MPQ factors ('constant', 'intermittent', 'muscular', VAS) and six *IPQ* factors (*psychological cause, physiological* cause, *external* cause, *timeline, consequence, control/cure*), making ten multiple tests. VAS was used in preference to PPI because of its better established validity and reliability (Pincus & Madland, 2000). Using Bonferroni's correction for 'familywise' type I error (Green et al, 1997, p.258), alpha must be less than .05/m for the most significant result, where m = the number of multiple tests, and less than .05/m-1 for the second most significant, etc. With ten multiple tests, therefore, the progression of significance will be: $p<.0050$, .0056, .0063, .0071, .0083, .01, .0125, .0167, .025, .05. Correlation tests were also performed with demographic and pain history factors, using a logarithmic transformation to normalise the 'duration' distribution.

Table 11.VI. Correlations for Entire Sample, MPQ factors and *IPQ factors* (*p<.05, **p<.01, ***p<.001).

	'Constant'	'Inter-mittent'	'Muscular'	VAS	*Psycho-logical*	*Physio-logical*	*External*	*Timeline*	*Con-sequence*	*Control / Cure*
'Constant'	/	.475***	.348***	.466***	.131	.261*	-.235*	.315**	.519***	-.184
'Inter-mittent'	.475***	/	.262**	.383***	.113	.109	-.303**	.250*	.381***	-.071
'Muscular'	.348***	.262**	/	.149	.097	.044	-.097	.238*	.143	.103
VAS	.466***	.383***	.149	/	-.163	.127	-.348***	.238*	.391***	-.241*
Psycho-logical	.131	.113	.097	-.163	/	.235*	.281**	.096	.201*	.209*
Physio-logical	.261*	.109	.044	.127	.235*	/	.140	.060	.303**	-.083
External	-.235*	-.303**	-.097	-.348***	.281**	.140	/	-.113	-.099	.189
Timeline	.315**	.250*	.238*	.238*	.096	.060	-.113	/	.360***	-.394***
Con-sequence	.519***	.381***	.143	.391***	.201*	.303**	-.099	.360***	/	-.197*
Control / Cure	-.184	-.071	.103	-.241*	.209*	-.083	.189	-.394***	-.197*	/

Psychological cause had no significant MPQ correlates but correlated with the other causal factors (*physiological*: R=.235, p=.023; *external*: .281, p=.008), and negatively with (years in full-time) education (-.282, p=.023) and frequency of pain episodes (-.222, p=.035). *Physiological* cause correlated negatively with frequency (-.217, p=.039), and positively with 'constant' pain (.261, p=.011), with *psychological* cause (.235, p=.023), and with *consequence* (.303, p=.003). *External* cause correlated significantly and negatively with both 'constant' and 'intermittent' pain (-.235, p=.027, and -.303, p=.004, respectively) and with pain intensity (VAS: -.348, p=.001).

Timeline correlated negatively with education (-.411, p=.000), and positively with all pain factors ('constant': .315, p=.002; 'intermittent': .250, p=.014; 'muscular': .237, p=.019; VAS: .238, p=.020). *Consequence* correlated with (log.) duration (.306, p=.002), education (negatively: -.357, p=.003), 'constant' pain (.519, p=.000), 'intermittent' pain (.381, p=.000) and pain intensity (.392, p=.000). *Control / Cure* correlated negatively with (log.) duration (-.227, p=.027) and pain intensity (-.241, p=.019).

11.IV.vi. 'Prediction' of Variance in Cognitions

Hierarchical Multiple Regression Analyses were then conducted to determine the contributions to variance in cognitions, with order of entry determined by the hypothetical schema (**Figure 9.I**, p.145). Stepwise Multiple Regression Analyses were also performed.

11.IV.vi.a. ***Psychological Causal Attributions***

In the hierarchical equation, a shorter education explained 6% of the variance in the attribution of pain to *psychological* causes (IPQ: **Table 11.VII.**). Lesser frequency of pain episodes explained a further 12%. Pain location did not contribute, and other cognitions added 7%, with 23% of the variance explained in total. The stepwise equation included frequency (12%), education (6%), and *external* causal attribution (6%).

Table 11.VII. Hierarchical Multiple Regression Analysis of *Psychological* Causal Attributions:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Education	-.284	.034	.06	.06
2	Frequency	-.358	.005	.18	.12
3	Location*	.046	.727	.16	-.02
4	*Physiological*	.199	.132	.23	.07
	External	.235	.061		
	Consequence	-.117	.448		
	Control/Cure	.078	.534		

Final regression equation (F 3.36, df 7, 48; p=.005)

*Location was scored as 1=TMD or 2=Headache.

11.IV.vi.b. ***Physiological Causal Attributions***

The hierarchical regression found lesser frequency of episodes to explain 4% of the variance, 'constant' pain character 5%, and other cognitions, notably perceived consequence, 4%. Location did not contribute (-1%), making a total prediction of only 12% (**Table 11.VIII.**). The stepwise equation included *consequence* and frequency only (10% and 4%, respectively).

Table 11.VIII. Hierarchical Multiple Regression Analysis of *Physiological* Causal Attributions:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Frequency	-.228	.035	.04	.04
2	'Constant'	.247	.020	.09	.05
3	Location	.070	.568	.08	-.01
4	*Psychological*	.106	.331	.12	.04
	Consequence	.244	.057		

Final regression equation (F 3.37, df 5, 80; p=.008)

11.IV.vi.c. ***External Causal Attributions***

In the hierarchical equation, pain intensity explained 10%; pain character (particularly non-intermittent) 1%; location 1%; and *psychological* causal attributions 8%; explaining a total 20% of the variance (**Table 11.IX.**). A stepwise equation included pain intensity (10%), *psychological* cause (5%), and 'intermittent' character (5%).

Table 11.IX. Hierarchical Multiple Regression Analysis of *External* Causal Attributions:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Intensity	-.328	.003	.10	.10
2	'Constant'	.029	.823	.11	.01
	'Intermittent'	-.221	.070		
3	Location	-.169	.199	.12	.01
4	*Psychological*	.304	.005	.20	.08

Final regression equation (F 4.94, df 5, 74; p=.000)

11.IV.vi.d. ***Timeline***

In the hierarchical analysis, education explained 19% of the variance in perceived *timeline*, intensity added 1%, whilst neither pain character nor location of pain contributed. Other cognitions, particularly *control/cure*, a negative correlate, contributed 24%, making a total 42% (**Table 11.X.**). The stepwise equation included education (19%), *control/cure* (20%), and 'muscular' character (3%; total 42%).

Table 11.X. Hierarchical Multiple Regression Analysis of Perceived *Timeline*:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Education	-.448	.000	.19	.19
2	Intensity	.167	.147	.20	.01
3	'Constant'	-.006	.964	.18	-.02
	'Intermittent'	.046	.740		
	'Muscular'	.114	.343		
	Location	.182	.298	.18	.00
4	*Consequence*	.218	.089	.42	.24
	Control/Cure	-.471	.000		

Final regression equation (F 6.61, df 8, 54; p=.000)

11.IV.vi.e. ***Consequence***

The hierarchical equation (**Table 11.XI.**) found less years in full-time education to explain 9% of the variance in perceived *consequence*; (log.) pain duration to explain a further 6%; pain intensity 13%; 'constant' pain character 12%; pain location 2%; and other cognitions (*physiological cause, timeline*) 5%. 47% of the variance was explained in total. The stepwise solution included 'constant' pain character and *timeline* only (37% and 8%, respectively).

Table 11.XI. Hierarchical Multiple Regression Analysis of Perceived *Consequence*:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Education	-.318	.013	.09	.09
2	Duration (log)	.284	.021	.15	.06
3	Intensity	.377	.001	.28	.13
4	'Constant'	.415	.002	.40	.12
	'Intermittent'	.072	.570		
5	Location	.207	.112	.42	.02
6	*Psychological*	-.085	.465	.47	.05
	Physiological	.185	.098		
	Timeline	.328	.016		
	Control/Cure	.138	.272		

Final regression equation (F 6.26, df 10, 49; p=.000)

11.IV.vi.f. *Control/Cure*

The hierarchical equation found shorter (log.) pain duration to explain 3% of the variance; intensity 3%; location 2%; and other cognitions (*psychological*, negative *timeline*) 20% (total: 28%, **Table 11.XII.**). The stepwise equation included *timeline* (16%), *psychological* (7%), and location (5%).

Table 11.XII. Hierarchical Multiple Regression Analysis of Perceived *Control/Cure*.

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Duration (log)	-.206	.057	.03	.03
2	Intensity	-.204	.059	.06	.03
3	Location	-.186	.100	.08	.02
4	*Psychological*	.291	.004	.28	.20
	Timeline	-.403	.000		
	Consequence	.087	.465		

Final regression equation (F 6.39, df 6, 79; p=.000)

11.V. DISCUSSION

The second study explored relations between pain character and pain beliefs, proposing that the former may drive the latter.

11.V.i. Causal Attributions

The causes of TMD and primary headache are unknown, and this lack of explanation is likely to contribute to individuals' distress consequent to the ongoing experience of pain (Magni et al, 1997; Madland et al, 2000). It was hypothesized that causal attributions would categorise into psychological and organic components, and that the headache group would preferentially endorse the former, and the TMD group the latter.

A previous principal component analysis of cardiac-related causal items in patients after myocardial infarction, a better understood condition perhaps, found three distinct factors of 'lifestyle', 'stress' and 'heredity' (Weinman et al, 1999). In contrast, this study has also found three distinct factors of *psychological, physiological* and *external* cause. These factors were clearly defined. It is interesting to note how the 'poor dental care' item loaded onto the external factor rather than the psychological factor, suggesting an interpretation of the care in question as that provided, or not, by others, i.e. dentists, rather than by the individuals themselves, an ambiguity which was not initially detected. The item was changed from 'poor medical care' in the original IPQ, the scale's authors having encouraged such adaptation.

The categorization of causal factors was thus more elaborate than that hypothesized (i.e. a three, rather than two, factor solution), though intriguingly similar to locus of control subscales: psychological / internal; physiological / chance; external /

powerful others. The insignificant distinction between groups in terms of causal belief fails to support the hypothesis.

11.V.ii. **Pain Character**

Regarding the MPQ, principal component analysis of both groups' scores on the pain descriptors was expected to derive a similar three-factor structure to that intended in the scale, i.e. intensity, sensory, and affective. In addition, the intensity subscale of the MPQ was expected to correlate with greater organic pain beliefs and lesser psychological pain beliefs for both groups, from the theory that the experience of intense pain would provoke beliefs in an underlying pathology, similar to the greater intensity of pain in an acute condition of known cause.

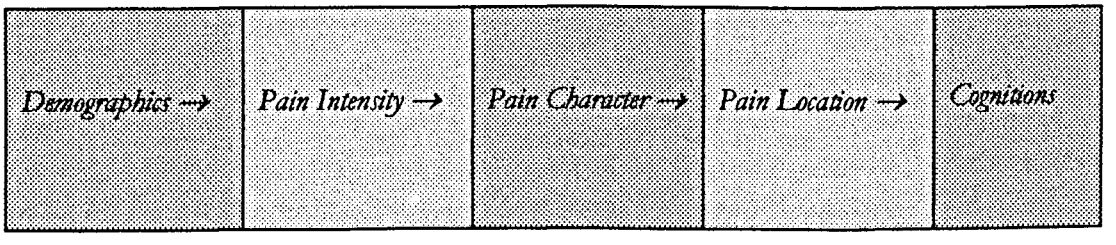
In the event, the PCA-derived factors differed markedly from Melzack's original formula of eleven 'sensory' and four 'affective' descriptors, and is perhaps closer to another solution derived from a Swedish translation of the scale, which found three factors in a group of fibromyalgia and rheumatoid arthritis patients: 'acute sensory', 'chronic sensory', and 'affective' (Burckhardt & Bjelle, 1994). Yet, in this study, there was no clean separation of sensory and affective descriptors, even when a 2-factor or 3-factor solution was forced.

Melzack's stipulation of 33%, as a cut-off prevalence of endorsement within a group to denote a descriptor's relevance to the pain condition in question, dictates that, from this study, 'stabbing', 'hot/burning', 'splitting', 'fearful', and 'punishing/cruel', are not considered, by TMD patients, to be appropriate descriptors of their symptoms. These results are comparable with those of Mongini et al (2000), who

found the most common descriptors in a TMD group to be: tiring (42%), troublesome (39%), nagging (37%), sore (35%), tender (26%), and aching (25%). Similarly, headache patients do not recognise 'gnawing' pain. Interestingly, 'splitting' was added to the MPQ as a 'key discriminative word for dental pain' (Melzack, 1987, p.192), suggesting that the TMD patients here were quite capable of distinguishing their symptoms from pain of dental origin. Otherwise, the wide range of descriptors endorsed reflects the anecdotally vague nature of these conditions. Although the headache group reported pain of greater duration and intensity than did the TMD group, the latter had more frequent symptoms, although this was not significant. The second and third sets of hypotheses were therefore not supported.

11.V.iii. **Prediction of Causal Attribution**

A correlational design cannot derive causal relationships, yet symptoms logically precede beliefs as to their cause, timeline, consequence, and control/cure, in as much as an individual need to experience a symptom before forming a definite set of beliefs about it. The hypothetical schema thus posits the variables in that order.



The relative failure of the included factors to predict causal attributions suggests that such appraisals involve subtler influences beyond this investigation. However, fewer

years in full-time education, and more pain-free days per month, did contribute significantly to the variance in attribution of pain to *psychological* causes. It may be that pain beliefs are influenced by aspects of higher and further education, or that socio-economic factors are involved, which were not assessed here. Attribution to *physiological* causes was also influenced by lower frequency, as well as by 'constant' pain character. This latter relation may suggest that individuals interpret throbbing, heavy, splitting, tiring, sickening, pain as arising from infection, diet, pollution, or hereditary factors. *External* causes were more likely to be blamed for less intense pain without 'intermittent' character; that is to say pain other than shooting, sharp or stabbing.

Within clinical research it is generally accepted, for both TMD and headache, that symptoms are initiated by local, albeit as yet unidentified, physiological phenomena, and then maintained and enhanced by psychosocial factors (see: RDC/TMD, ICD-10). In this study, the equivalent endorsement of psychological, physiological and external causes suggests that patients too are aware of an aetiological synergy of all three.

11.V.iv. **Prediction of Other Pain-Related Cognitions**

Anticipated endurance of symptoms (timeline) was principally associated with a shorter experience of full-time education, and with a lack of belief in control/cure, the latter being logical. Perceived consequence of pain was similarly influenced by education level, as well as by duration, intensity and 'constant' pain character; whilst belief in *control/cure* was partially explained by shorter duration, lesser intensity, and location, with TMD perceived as more controllable/curable than was headache.

Thus, in classifying pain according to its character, constant pain is more likely than pain of other character to be blamed on *physiological* causes, and to be perceived as consequential.

Pain location, namely TMD or headache, is a relatively insignificant predictor of pain beliefs, after controlling for duration, frequency, intensity and character, supporting the generalisation of the above relations across both pain conditions.

The following chapter describes the longitudinal part of this TMD / Headache comparison, and examines the relationships between cognitions and outcome.

Chapter 12. TEMPOROMANDIBULAR DISORDER AND HEADACHE: COGNITIONS AND OUTCOME

12.I AIM

Having considered what aspects of the pain experience may drive cognitions, a longitudinal investigation was now conducted into the influence of those cognitions on change in pain, disability and distress. In addition to those factors described in the previous chapter, putative mediating factors were assessed, including coping style, anger style and alexithymia.

12.II. MEASURES

Age, Gender and Demographic Characteristics including marital status, occupation and years in full-time education.

Pain Duration (months) **and Frequency** (days with pain during preceding month)

Illness Perceptions Questionnaire (IPQ) (Weinman et al, 1996; pp. 101, 139)

McGill Pain Questionnaire (SF-MPQ) (Melzack, 1987; p.137).

Disability Checklist (p.136)

Coping Strategies Questionnaire (CSQ) (Rosenstiel & Keefe, 1983; pp.86, 140).

Hospital Anxiety and Depression scale (HAD) (Zigmond & Snaith, 1983; pp. 117, 137).

Perceived Stress Scale (PSS-10) (Cohen et al, 1983; p.141)

State-Trait Anger Expression Inventory (STAXI) (Spielberger, 1988; p. 141).

Toronto Alexithymia Scale (TAS-20) (Bagby et al, 1994; p.141).

Analgesic (non-steroidals, paracetamol) **and Prophylactic** (tricyclics, serotonin antagonists, beta-blockers) **Medication Use** ("yes" or "no")

Clinical Signs (based on RDC/TMD; **Appendix IV**).

12.III. PROCEDURE

The fifty-one TMD and fifty-one headache patients completed the questionnaires (**Appendix II**) at the same time as the scales described in the previous chapter. As stated, all participants were aged 18 to 70 years and English speakers, those suffering from other painful chronic illness were excluded, informed consent and ethical approval was obtained. TMD patients were recruited from new referrals to the Oral & Maxillofacial Surgery departments of: University College Hospital, London; the Royal Free Hospital, London; the Whittington Hospital, London; Chase Farm Hospital, Enfield, Middlesex; and the Royal Berkshire Hospital, Reading, Berkshire. Participants satisfied the research diagnostic criteria (RDC/TMD, Dworkin & LeResche, 1992). Headache subjects were recruited from the Neurology departments of Wexham Park Hospital, Slough, Berkshire, and the Royal Berkshire Hospital, Reading, Berkshire. Patients were invited to complete the questionnaire at the time of their initial outpatient appointment.

Management largely involved reassurance as to the benign nature of both conditions, which was confirmed radiographically (no lesions were detected). Patients were introduced to the idea of a relationship between pain and psychological stress, and prophylactic medication (usually with a tricyclic antidepressant) was recommended. The decision whether to take medication was left to the patient.

A six-month follow-up questionnaire, identical to the initial one, was posted to the TMD and headache patients, of whom 76% and 65% replied, respectively. Six months, though arbitrary, is a frequently used time period for longitudinal study (for examples: Turner et al, 1995, Turk et al, 1996), and has the semantic advantage that any subject still suffering symptoms at follow-up is, by definition, a chronic pain patient, despite the variable duration of symptoms within each group. The attrition rates are relatively high – 10% would be more acceptable. However, such drop-out must be expected when no additional treatment is being offered. The questionnaires are long, and participants often fail to see any incentive in repeating identical questionnaires. In addition, it may have appeared to some headache sufferers that the research was primarily directed at facial pain, further compromising their continuing cooperation.

12.IV. RESULTS

12.IV.i. Inter-Group Differences at T_1

Table 12.I. shows the group means and significant differences at T_1 (initial specialist consultation). There were significant inter-group differences on several variables. Headache patients reported longer duration of their condition than did TMD patients (mean 72, SD 115 vs. 30, SD 57; $p<0.05$), greater pain intensity over the preceding month on both visual analogue (66, SD 24 vs. 48, SD 26; $p<0.01$) and verbal rating (3.3, SD 1.1 vs. 2.3, SD 1.1; $p<0.01$), and more depressed mood (7.6, SD 4.9 vs. 4.2, SD 3.9; $p<0.01$). Differences were also detected on TAS, IPQ and CSQ factors. Headache patients were more alexithymic than TMD patients on TAS factors 2 "difficulty describing feelings" (13.9, SD 4.5 vs. 11.0, SD 4.2; $p<0.01$) and 3 "externally-oriented thinking" (21.5, SD 4.2 vs. 18.0, SD 4.7; $p<0.01$). Headache patients considered their condition to be of greater consequence (37.0, SD 7.4 vs. 28.8, SD 8.0; $p<0.01$) and more puzzling (4.0, SD 1.1 vs. 3.4, SD 1.1; $p<0.01$) than did TMD patients. Headache patients also catastrophised more than TMD patients about their pain (14.0, SD 10.3 vs. 7.7 SD 7.9; $p<0.01$), whilst TMD patients were more inclined to ignore sensations (15.8, SD 7.6 vs. 11.2, SD 8.3; $p<0.01$) and perceive greater control over pain (4.0, SD 3.0 vs. 2.0, SD 2.6; $p<0.01$). Inter-group differences in signs and symptoms of jaw function were unsurprising. Note that the majority of symptoms making up the disability score was jaw-related (18 out 27).

Analyses of Co-Variance (ANCOVA's) were then performed to establish whether the inter-group differences in cognitions could simply be attributed to the observed differences in pain intensity and duration. The answer was 'no' for: TAS factors 2 and 3; IPQ consequence; and CSQ 'control over pain'. Similarly, the inter-group

difference in depressed mood was not explained by the differences in pain intensity and duration alone. These differences ($p<.01$) can therefore be attributed to the different conditions themselves.

12.IV.ii. **Inter-Group Differences at T_2**

For the TMD group, the followed-up patients (N=39) differed significantly from the drop-out group (N=12) on CSQ factor 'perceived control over pain' (mean 4.7, SD 3.0 vs. 1.9, 2.3). For the headache patients there were no significant differences between followed-up (N=33) and drop-out groups (N=18).

Table 12.II. shows the group means at T_2 (six months post-consultation). There was one significant difference ($p<0.01$): attribution to physiological causes was greater in the headache group (8.9, SD 3.3, vs. 6.9, 2.7).

12.IV.iii. **Inter-Group Differences in Changes (T_2 - T_1)**

Changes for the better in pain, anxious mood and depressed mood, were significantly greater for the headache group than for the TMD group (**Table 12.III.**). The headache group's VAS score for pain over the preceding month improved by 24 points (SD 34), compared with a 6 point improvement in the TMD group (SD 24, $p<.05$); anxious mood improved by 1.5 points (SD 3.3) in the headache group, compared with a worsening by 0.5 points (SD 3.3, $p<.05$) in the TMD group; whilst depressed mood improved by 2.7 points (SD 4.0), compared with a worsening by 0.3 points (SD 3.3, $p<.01$). Changes in cognitions and coping strategies did not differ significantly between the groups.

Table 12.I. Group means (and SDs); and significant differences: **p<0.01, *p<0.05

At T1:	**TMD**	**HEAD**	range	p
N	51	51		
EXAM - unassisted jaw opening (mm)	38 (11)	44 (7)	20 - 62	**
asisted jaw opening (mm)	39 (10)	44 (7)	20 - 62	*
jaw sounds (on palpation)	0.7 (0.5)	0.0 (0.2)	0 - 1	**
arthralgia (on palpation)	0.6 (0.5)	0.2 (0.4)	0 - 1	**
myalgia (on palpation)	0.5 (0.5)	0.2 (0.4)	0 - 1	**
STAXI - anger control	14.7 (5.4)	14.6 (5.4)	0 - 24	
anger expression	6.6 (3.6)	6.2 (3.9)	0 - 24	
anger repression	7.9 (3.7)	9.1 (4.3)	0 - 24	
TAS - alexithymia	45.5 (12.0)	54.1 (12.8)	20 - 100	**
1. difficulty identifying feelings	16.2 (6.7)	18.7 (7.2)	7 - 35	
2. difficulty describing feelings	11.1 (4.2)	13.9 (4.5)	5 - 25	**
3. externally-oriented thinking	18.1 (4.6)	21.6 (4.2)	8 - 40	**

At T1:	TMD	HEAD	range	p
IPQ –				
psychological	7.9 (3.2)	8.3 (2.8)	3 – 15	
physiological	7.6 (3.3)	9.0 (3.0)	4 – 20	*
external	7.7 (2.3)	6.8 (2.4)	3 - 15	
timeline	9.2 (2.4)	9.8 (2.1)	3 - 15	
consequence	18.3 (5.1)	23.1 (4.8)	7 - 35	**
control/cure	19.8 (2.7)	18.4 (2.5)	6 - 30	*
CSQ -				
diverting attention	8.1 (7.6)	7.9 (8.9)	0 - 36	
increasing behavioural activities	12.8 (10.7)	9.8 (6.1)	0 - 36	
ignoring sensations	15.9 (7.7)	11.2 (8.3)	0 - 36	
reinterpreting sensatioons	5.8 (6.6)	4.8 (5.4)	0 - 36	
coping self-statements	20.7 (7.5)	16.5 (10.0)	0 - 36	*
praying / hoping	10.9 (8.7)	15.0 (10.7)	0 - 36	*
catastrophising	7.8 (8.0)	14.0 (10.2)	0 - 36	**
control	4.0 (3.0)	2.0 (2.6)	0 - 12	**
PSS – stress	16.8 (8.5)	18.2 (7.1)	0 - 40	

At T1:	TMD	HEAD	range	p
Pain -				
past, VAS	48 (27)	66 (24)	0 - 100	**
past, PPI	2.3 (1.1)	3.3 (1.1)	0 - 5	**
current, VAS	29 (24)	39 (29)	0 - 100	
current, PPI	1.5 (1.1)	1.7 (1.3)	0 - 5	
HAD -				
anxiety	7.6 (5.0)	9.4 (4.3)	0 - 21	
depression	4.1 (3.9)	7.6 (4.9)	0 - 21	**
Disability	22.1 (9.5)	18.0 (8.0)	0 - 81	*

Table 12.II. Group means (and SDs); and significant differences: **p<0.01, *p<0.05

At T2:	**TMD**	**HEAD**	range	
N	39	33		
IPQ – psychological	8.0 (3.0)	8.5 (2.9)	3 - 15	
physiological	6.9 (2.7)	8.9 (3.3)	4 – 20	**
external	7.7 (1.9)	7.3 (2.2)	3 – 15	
timeline	9.0 (2.3)	9.3 (2.2)	3 – 15	
consequence	20.4 (5.2)	23.1 (6.2)	7 – 35	
control/cure	19.1 (3.6)	18.1 (4.1)	6 - 30	
CSQ - diverting attention	7.9 (7.8)	7.8 (6.5)	0 - 36	
increasing behavioural activities	11.3 (8.8)	9.3 (6.1)	0 - 36	
ignoring sensations	15.3 (8.2)	12.4 (7.7)	0 - 36	
reinterpreting sensations	6.3 (5.7)	4.2 (4.9)	0 - 36	
coping self-statements	16.6 (8.1)	18.2 (8.5)	0 - 36	
praying / hoping	7.8 (8.7)	11.2 (9.6)	0 - 36	
catastrophising	8.0 (9.6)	12.0 (10.6)	0 - 36	
Control	4.3 (2.5)	3.2 (3.3)	0 - 12	

At T2:	**TMD**	**HEAD**	range	
PSS -stress	16.8 (6.6)	17.4 (6.6)	0 - 40	
Pain -				
past, VAS	38 (27)	45 (27)	0 - 100	
past, PPI	2.0 (1.4)	2.5 (1.3)	0 - 5	
current, VAS	27 (27)	28 (29)	0 - 100	
current, PPI	1.1 (1.1)	1.2 (1.3)	0 - 5	
HAD -				
anxiety	8.5 (4.3)	7.5 (3.8)	0 - 21	
depression	4.3 (3.9)	3.9 (3.5)	0 - 21	
Disability	20.4 (10.7)	15.5 (7.7)	0 - 81	*

Table 12.III. Group mean changes over time (and SDs); and significant differences: **p<0.01, *p<0.05

Change (X_{T1} minus X_{T2}):	**TMD**	**HEAD**	p
Pain – past, VAS	6 (25)	24 (34)	*
Disability	1.2 (6.6)	2.2 (6.5)	
HAD – anxiety	-0.5 (3.3)	1.5 (3.3)	*
depression	-0.3 (3.3)	2.7 (4.0)	**
IPQ – psychological	-0.1	-0.2	
physiological	0.7	0.1	
external	0.0	-0.5	
timeline	0.2 (2.5)	1.0 (1.9)	
consequence	-2.2 (4.0)	-0.5 (5.4)	
control/cure	0.8 (3.1)	-0.1 (3.4)	
CSQ - diverting attention	0.5 (5.4)	0.6 (8.5)	
increasing behavioural activities	1.5 (6.2)	1.9 (8.2)	
ignoring sensations	-0.5 (8.3)	0.5 (9.9)	
reinterpreting sensations	-0.3 (5.4)	0.4 (6.1)	
coping self-statements	4.7 (8.7)	-0.2 (9.1)	
praying / hoping	2.7 (8.9)	1.8 (6.0)	
catastrophising	-0.9 (6.3)	1.1 (8.0)	
control	0.5 (3.5)	-0.7 (3.9)	

12.IV.iv. **Correlation of Changes in Cognitions and Outcome Measures (Table 12.IV.)**

Bonferroni's correction for familywise type I error (p.179) dictates that, for fifteen multiple tests, the progression of significance will be: $p<.003$, .004, .004,.... Change in pain correlated significantly with change in catastrophising (CSQ: .472, $p<.01$). Change in disability correlated with change in timeline (.372) and consequence (.365), as well as with change in catastrophising (.367). Change in anxious mood correlated with change in timeline (.509) and in catastrophising (.391). Change in depressed mood correlated with change in consequence (.358), and, negatively, in coping self-statements (-.359).

12.IV.v. **Prediction at T_1 of Dependent Variables at T_2**

Correlation analyses were performed to establish which variables at T_1 predicted dependent variables at T_2 Significant correlates were then entered into hierarchical multiple regression equations, according to the schema (**Figure 12.I.**), to determine the relative predictive power. The corresponding 'dependent' variable at T_1 was entered first in each equation to control for initial differences. Pain location (face or head) was entered only if a significant difference in means was observed.

Table 12.IV. Correlations between Changes in Cognitions and Outcomes (Changes in DV's; *p<.05, **p<.01):

	Δ pain	Δ dis'y	Δ anx	Δ dep	Δ time	Δ cons	Δ cure	Δ DA	Δ IBA	Δ RS	Δ CSS	Δ IS	Δ PH	Δ cat	Δ cont
Δ pain	/	.280*	.332**	.316*	.264*	.290*	-.199	.066	-.029	.136	-.080	-.038	.173	.472**	-.149
Δ dis'y	.280*	/	.326**	.305*	.372**	.365**	-.298*	.304*	.049	.084	-.038	-.078	.091	.367**	-.291*
Δ anx	.332**	.326**	/	.423	.509*	.284*	-.214	-.031	.155	.042	-.156	.074	.198	.391**	-.265*
Δ dep	.316*	.305*	.423**	/	.256*	.358**	-.260*	-.216	-.200	-.041	-.359**	-.061	-.066	.262*	-.255
Δ time	.264*	.372**	.509**	.256	/	.396**	-.424**	.036	.181	.032	-.140	-.055	.023	.168	-.362**
Δ cons	.290*	.365**	.284*	.358	.396**	/	-.410**	.026	-.127	-.116	-.329**	-.298*	.074	.330**	-.420**
Δ cure	-.199	-.298*	-.214	-.260	-.424**	-.410**	/	-.045	-.018	-.212	.024	.073	.174	-.140	.353**
Δ DA	.066	.304*	-.031	-.216	.036	.026	-.045	/	.540**	.365**	.370**	.298*	.191	.133	-.069
Δ IBA	-.029	.049	.155	-.200	.181	-.127	-.018	.540**	/	.467**	.435**	.240*	.453**	.124	.039
Δ RS	.136	.084	.042	-.041	.032	-.116	-.212	.365**	.467**	/	.419**	.241	.212	.092	-.027
Δ CSS	-.080	-.038	-.156	-.359	-.140	-.329**	.024	.370**	.435**	.419**	/	.451**	.158	-.076	.342**
Δ IS	-.038	-.078	.074	-.061	-.055	-.298*	.073	.298*	.240	.241	.451**	/	.098	-.026	.095
Δ PH	.173	.091	.198	-.066	.023	.074	.174	.191	.453**	.212	.158	.098	/	.379**	.196
Δ cat	.472**	.367**	.391**	.262*	.168	.330**	-.140	.133	.124	.092	-.076	-.026	.379	/	.149
Δ cont	-.149	-.291	-.265*	-.255	-.362**	-.420**	.353**	-.069	.039	-.027	.342**	.095	.196	-.149	/

12.IV.v.a. ***Current Pain at T_2***

Current pain at T_2 was predicted by: current pain at T_1 (11%), perceived control/cure (negative) and consequence (5%), and disability (7%, total: 23%, **Table 12.V.**).

Table 12.V.

Hierarchical Multiple Regression analysis of variables at T_1 on Current Pain (VAS) at T_2, n=61:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Current Pain VAS	.356	.005	.11	.11
2	Control/Cure (IPQ)	-.225	.069	.16	.05
	Consequence (IPQ)	.152	.243		
3	Catastro-phising (CSQ)	.110	.500	.16	.00
4	Disability	.294	.013	.23	.07

Final regression equation (F 4.67, df 5, 56; p=.001)

12.IV.v.b. ***Disability***

Disability at T_2 was predicted by: disability at T_1 (55%). Other variables did not contribute to the final equation (**Table 12.VI.**).

Table 12.VI.

Hierarchical Multiple Regression analysis of variables at T_1 on Disability at T_2, n=64:

step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Disability	.748	.000	.55	.55
2	Stress (PSS)	.073	.442	.55	.00
3	Timeline (IPQ)	.057	.527	.54	-.01
4	Anger repression (STAXI)	.023	.827	.53	-.01
	Increasing Behavioural Activities (CSQ)	.066	.489		

Final regression equation (F 15.67; df 5, 59; p=.000)

12.IV.v.c. ***Anxious mood at T_2***

Anxious mood at T_2 was predicted by: anxious mood at T_1 (51%), and stress (1%, **Table 12.VII.**). Perceived psychological cause, anger repression, catastrophising, and alexithymia did not contribute.

Table 12.VII.

Hierarchical Multiple Regression analysis of variables at T_1 on Anxious Mood at T_2, n=60:

Step	variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Anxious mood (HAD)	.626	.000	.51	.51
	Depressed mood (HAD)	.136	.263		
2	Stress (PSS)	.235	.082	.51	.01
3	Psychological (IPQ)	.090	.379	.51	.00
4	Catastro-phising (CSQ)	.137	.175	.51	.00
	Anger repression (STAXI)	.076	.514		
	Alexithymia (TAS 1)	-.109	.412		

Final regression equation (F 10.43, df 7, 53; p=.000)

12.IV.v.d. ***Depressed mood at T_2***

Depressed mood at T_2 was predicted by: depressed mood at T_1 (27%), and perceived psychological cause and consequence (2%, total: 26%, **Table 12.VIII.**). Stress, anger repression, catastrophising, and alexithymia were non-contributory.

Table 12.VIII. Hierarchical Multiple Regression analysis of variables at T_1 on Depressed Mood at T_2, n=60:

Step	Variables	Beta	significance T	cumulative adjusted R^2	incremental R^2
1	Depressed mood (HAD)	.386	.010	.27	.27
	Anxious mood (HAD)	.209	.158		
2	Stress (PSS)	-.006	.969	.26	-.01
3	Psychological (IPQ)	.195	.121	.28	.02
	Consequence (IPQ)	.142	.266		
4	Catastro-phising (CSQ)	.135	.341	.26	-.02
	Anger repression (STAXI)	.058	.691		
	Alexithymia (TAS 1)	-.052	.752		

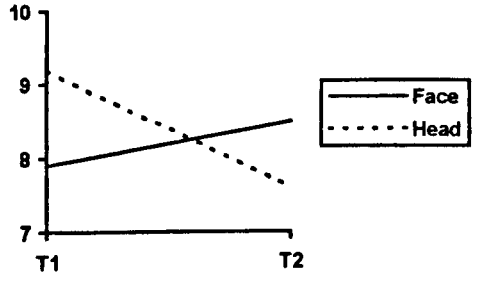
Final regression equation (F 3.62, df 8, 52; p=.002)

12. IV.vi. **Inter-Group Differences in Outcome**

Repeated Measure Analyses of Variance (ANOVA's) were conducted, in order to determine whether changes over time differed significantly between groups (**Figure 12.II.**). Changes in four of the dependent variables differed significantly between groups: anxious mood and current pain ($p<0.05$), depressed mood and pain over the preceding month ($p<0.01$). Anxious mood decreased in the headache group but actually increased in the TMD group, whilst depressed mood decreased in the headache group to a level comparable to that of the TMD group. Past pain (preceding month VAS) decreased in both groups but more so for the headache patients. Current pain (VAS) decreased in the headache group only.

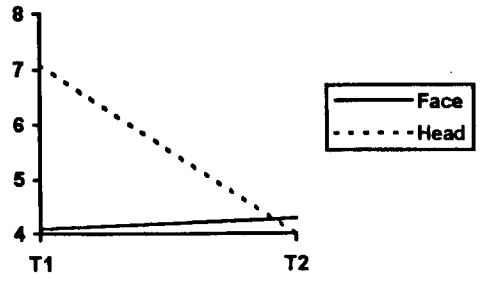
Figure 12.II: ANOVA's (repeated measures, significance of group-factor interaction shown)

Anxiety (HAD)



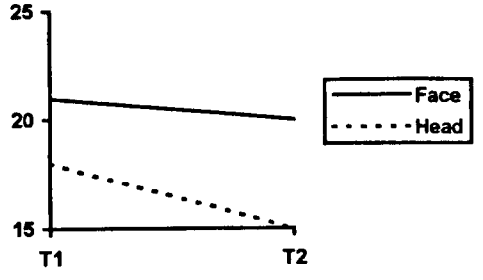
group: F=.001, sig=.980
time: F=1.43, sig=.236
time x group: F=6.47, sig=.013
df, error=65

Depression (HAD)



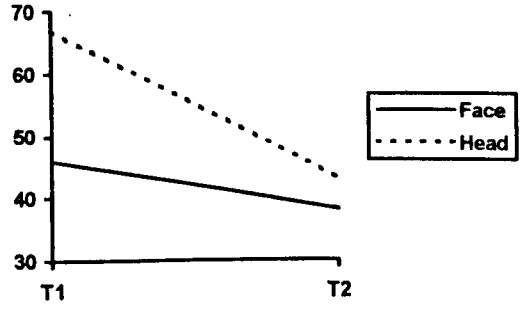
group: F=1.78, sig=.187
time: F=7.33, sig=.009
time x group: F=11.4, sig=.001
df, error= 65

Disability



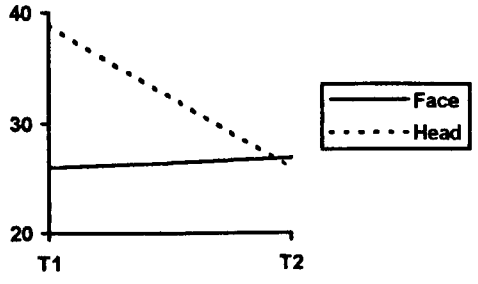
group: F=6.05, sig=.016
time: F=4.56, sig=.036
time x group: F=.360, sig=.551
df, error= 68

Past Pain (VAS)



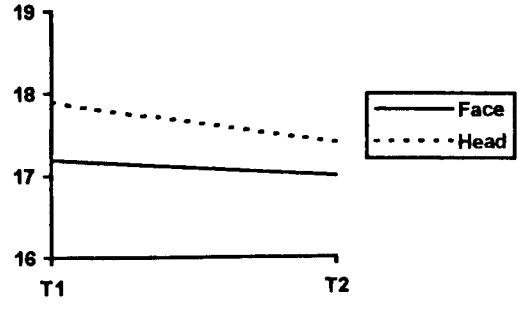
group: F=5.50, sig=.022
time: F=16.7, sig=.015
time x group: F=6.30, sig=.015
df, error= 66

Current Pain (VAS)



group: F=.377, sig=.541
time: F=4.67, sig=.034
time x group: F=4.67, sig=.034
df, error= 63

Stress (PSS)



group: F=.161, sig=.689
time: F=.279, sig=.599
time x group: F=.106, sig=.746
df, error= 66

12.IV.vii. **Association of Changes with Treatment Modality**

The only significant effect associated with pharmaceutical treatment was on anxiety. Patients taking prophylactic medication (tricyclic antidepressants, etc.) had a greater reduction in anxiety, even controlling for initially higher anxiety, than those not taking such medication (significance of F = 0.002).

The association of reduction in anxious mood with prophylactic medication (largely tricyclic antidepressants) might have accounted for some of the headache group's improvement. Prophylactic medication was more prevalent in the headache group than in the TMD group (**Table 20.IX**).

Table 12.IX. Prevalence of Prophylactic Medication in the Two Groups (%).

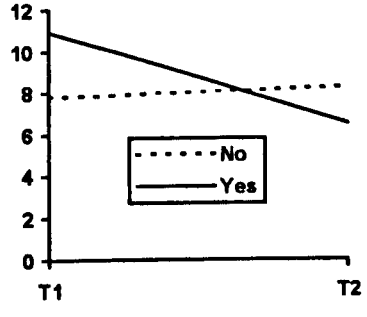
	TMD Group	Headache Group
Prophylactic Medication at T1	14 %	28 %
Prophylactic Medication at T2	12 %	20 %

Figure 12.III. ANOVA's, repeated measures

Within-subjects factor: Time;

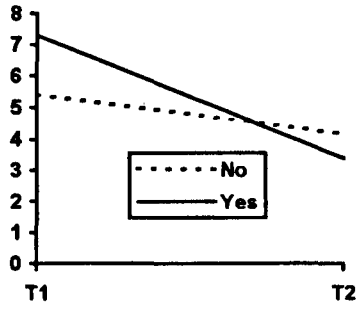
Between-subjects factor: Medication at T_1 (prophylaxis: no/yes).

Anxiety



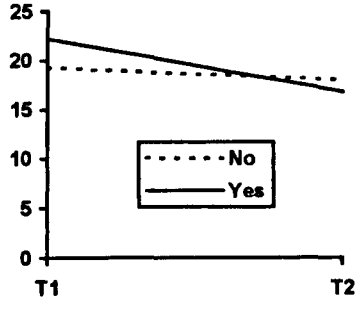
medn: F=.065, sig=.800
time: F=6.70, sig=.012
time x medn: F=9.00, sig=.004
df, error= 65

Depression



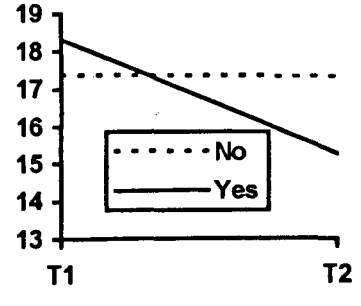
medn: F=.110, sig=.741
time: F=5.26, sig=.025
time x medn: F=.822, sig=.368
df, error= 65

Disability



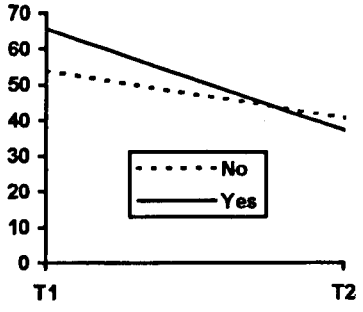
medn: F=.256, sig=.614
time: F=7.47, sig=.008
time x medn: F=2.94, sig=.091
df, error= 68

Stress



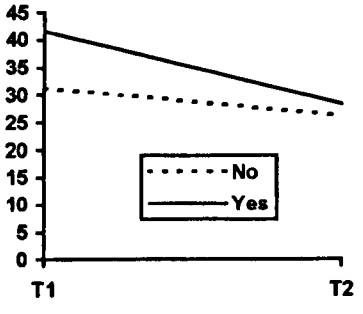
medn: F=.363, sig=.549
time: F=.950, sig=.333
time x medn: F=.850, sig=.360
df, error= 66

Past Pain VAS



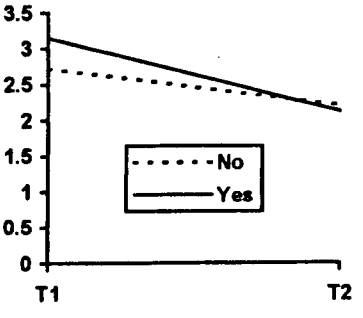
medn:	F=1.07, sig=.305
time:	**F=18.3, sig=.000**
time x medn:	F=4.37, sig=.041
df, error= 64	

Current Pain VAS



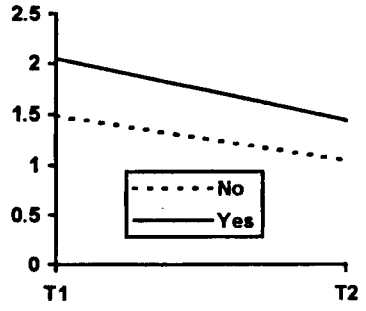
medn:	F=.975, sig=.327
time:	F=5.18, sig=.026
time x medn:	F=1.59, sig=.212
df, error= 63	

Past Pain VRI



medn:	F=.348, sig=.557
time:	F=6.48, sig=.013
time x medn:	F=1.09, sig=.299
df, error= 69	

Current Pain VRI



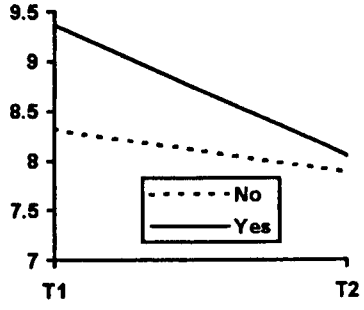
medn:	F=2.46, sig=.121
time:	**F=9.49, sig=.003**
time x medn:	F=.000, sig=.985
df, error= 65	

ANOVA's, repeated measures.

Within-subjects factor: Time;

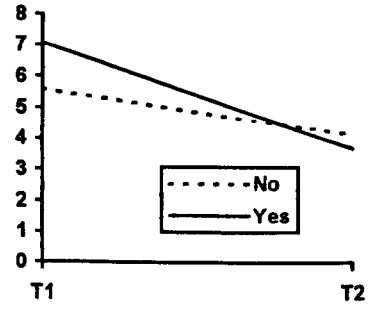
Between-subjects factor: Medication at T_2 (prophylaxis: no/yes).

Anxiety



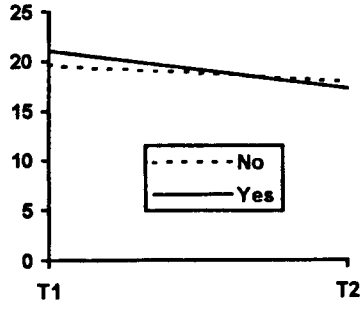
medn: F=.330, sig=.568
time: F=1.27, sig=.264
time x medn: F=.469, sig=.496
df, error= 65

Depression



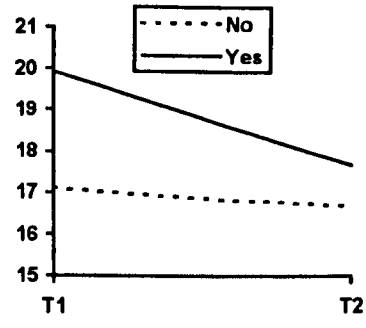
medn: F=.796, sig=.376
time: F=10.7, sig=.002
time x medn: F=5.93, sig=.018
df, error= 65

Disability



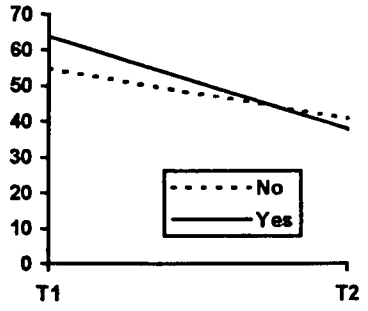
medn: F=.000, sig=.996
time: F=6.67, sig=.012
time x medn: F=2.19, sig=.144
df, error= 68

Stress



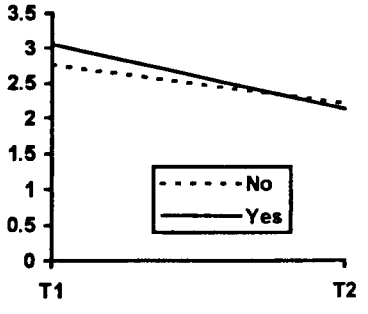
medn: F=1.30, sig=.258
time: F=1.31, sig=.256
time x medn: F=1.72, sig=.195
df, error= 66

Past Pain VAS



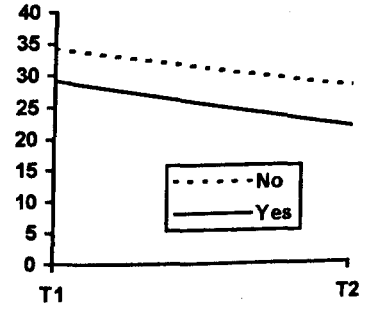
medn: F=.571, sig=.453
time: F=17.4, sig=.000
time x medn: F=3.58, sig=.063
df, error= 64

Past Pain VRI



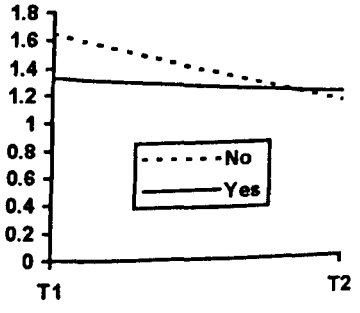
medn: F=.540, sig=.465
time: F=9.65, sig=.003
time x medn: F=3.74, sig=.037
df, error= 69

Current Pain VAS



medn: F=.479, sig=.491
time: F=2.91, sig=.093
time x medn: F=.057, sig=.813
df, error= 63

Current Pain VRI



medn: F=.344, sig=.559
time: F=6.69, sig=.012
time x medn: F=1.23, sig=.272
df, error= 65

12.V. DISCUSSION

12.V.i. **Initial Group Differences**

The longitudinal study considered how cognitions relate to pain, disability and distress over time. The inter-group differences suggested that, by the time of the initial specialist consultation, the ongoing experience of chronic headache is essentially *worse* than that of TMD. Despite the initial disparities in pain intensity and duration, these did not account for the differences in perceived consequence, difficulty in description, externally-oriented thinking, and lack of control, all of which were greater for the headache group.

12.V.ii. **Final Group Differences**

Six months after their initial hospital consultation, the headache group showed greater improvement than did the TMD group, in terms of pain and distress, suggesting their management to be the more effective.

TMD and headache groups had been expected to show modest and comparable improvement in the primary outcome measures of pain, disability and distress, over six months, in line with other studies (Turner et al, 1995; ter Kuile et al, 1995; see p.154). This hypothesis was not supported for the TMD group. Over the same period, the differences between the groups disappeared, despite the dropping out from the TMD group of several individuals with low perceived pain control.

The headache group may also have experienced greater reassurance from the consultation and management than did the TMD group, particularly with regard to cancer-phobia, as there is anecdotal evidence for such a fear in headache sufferers, which is addressed with reassurance and radiography. Unfortunately the IPQ scale

did not include any cancer-related items, and the only inter-group difference in causal attribution was a persistent greater endorsement of diet in the headache group than in the TMD group, which approached significance (T1: p=.014; T2: p=.018).

The beneficial effect of prophylaxis with tricyclics was only apparent in reducing anxiety. Since such medication was recommended to all at consultation, the decision whether to take it or not is likely to have been influenced by cognitive factors, such as attitudes to medication, and to long-term psychotropic medication in particular. Evidently this was a vague measure of medical management, since the study was not primarily concerned with treatment but rather with the relations between cognitions and outcome. Rather than demonstrating an effect of medication, it might better be considered a factor in determining whether an individual is predisposed toward medical management of their condition.

12.V.iii. **Prediction of Outcome**

The hierarchical multiple regression equations confirm that the best predictors, across the entire sample, of disability and distress over the following six months, were disability and distress at the time of initial consultation. Initial cognitions made no significant contribution. Only pain intensity could be partially predicted by a cognitive factor, namely lack of perceived controllability / curability (IPQ, 5%, p=.07), and the total predictive capacity for the whole model only reached 23% for this variable.

The remaining hypotheses were therefore unsupported:

9. Improvements in pain, disability and distress were expected to be associated with reductions in perceived consequence and time-line (i.e. persistence), increased endorsement of active coping strategies, and reductions in passive coping strategies.

10. Improvement over six months was expected to be predicted by belief in psychological cause of pain, and persistence of symptoms predicted by organic causal belief in the absence of any findings of pathology.

11. Alexithymia and repressive anger style were expected to negatively influence improvement in primary outcome, in both groups.

12. **Figure 9.III.** shows a putative schema, to be demonstrated in hierarchical multiple regression analyses for the primary outcomes.

Experience→	*Stressors→*	*Assessment→*	*Coping→*	*Reaction*
Demographics	Past pain Stress	Illness perceptions	Alexithymia Anger style Coping strategies	Current pain Disability Mood

These results must be seen in the light of the overall lack of improvement, particularly for the TMD group. The reason that dependent variables at T_1 were largely predictive of the same variables at T_2 is likely to be that these variables hardly changed over the time period assessed, and that there was therefore little else to predict. In this respect, the correlations of change, or lack of change, in cognitions

and outcome are perhaps more enlightening. Perceived permanence ('timeline', IPQ) correlated significantly with disability and anxious mood; perceived consequence (IPQ) with disability and depressed mood; and catastrophising (CSQ) with pain, disability, and anxious mood.

Whatever the reasons, the recalcitrance of the TMD group's pain, disability and distress indicates the need for a change in approach to management.

Chapter 13. A PSYCHO-EDUCATION PROGRAMME FOR TMD

13.I. INTRODUCTION AND AIM

An attempt was made to draw together the findings of the literature reviewed above and the results of the questionnaire-based studies into a psycho-education programme for TMD patients. The aim of this next project was to develop and pilot a simple, cost-effective, evidence-based management programme for TMD, using CD-ROM. A comparison group received adjunctive relaxation training, known to be effective in the management of this disorder.

13.II. EDUCATION AND SELF-MANAGEMENT IN CHRONIC PAIN

13.II.i. Cognitive Behavioural Therapy

Cognitive Behavioural Therapy (CBT) aims to enable people to better manage their difficulties by applying empirically researched principles of thoughts, feelings and behaviours. These principles translate into practical strategies, which can lead to changes in subjective and objective thoughts, feelings and behavioural states. Interventions include: goal setting; challenging negative automatic thoughts; relaxation and breathing exercises; cognitive visualization exercises; behavioural coping strategies; stress management and assertion skills.

CBT is an effective approach to the management of many chronic pain conditions, despite the difficulties in conducting blind controlled trials (Morley et al, 1999). CBT has been found to be effective in reducing pain and disability in TMD, particularly in combination with other treatment modalities, such as medication with fluoxetine (Harrison et al, 1997) and biofeedback (Gardea et al, 2001).

Cognitive therapy produced continued improvements in pain up to six months after a six week treatment programme consisting of an intra-oral appliance and stress management with biofeedback, compared to the same programme with non-directive supportive counseling, in a group of TMD patients classified as 'dysfunctional' according to the MPI (Turk et al, 1996). However, the majority of TMD patients is not dysfunctional, and might therefore respond to a simpler approach based on education and self-management.

13.II.ii. **Education Programmes**

There is very little literature on the topic of education programmes for chronic pain, despite their importance in the care of patients with, for example, rheumatic disease (Burckhardt et al, 1994). Much work has been conducted by Lorig and her colleagues with rheumatoid arthritis sufferers. Their Arthritis Self-Management Program (ASMP) was found to produce long-term benefits in terms of reduced pain ratings and arthritis-related physician visits, saving approximately £400 per patient over 4 years (Lorig et al, 1993). Other studies have established benefits from telephone- and mail-delivered self-management programmes for arthritis (Maisiak et al, 1994; Fries et al, 1997).

In a randomised, controlled trial of education and physical training for women with fibromyalgia, of which TMD may be a variant, the education programme was found to enhance self-efficacy, although changes in disability and distress were more modest (Burckhardt et al, 1994). These results are encouraging when one considers the potential effect of enhanced self-efficacy on perceived control over pain. A

Canadian group of mixed idiopathic chronic pain patients, assigned to a community-based psycho-education programme modified from the Arthritis Self-Management Programme, made significant short-term improvements in pain, dependency, vitality, aspects of role functioning, life satisfaction and in self-efficacy and resourcefulness, compared to a waiting-list control group (LeFort et al, 1997).

An evidence-based educational booklet for back pain, published in the UK, has been found to produce a positive shift in beliefs and a significant reduction in self-reported disability, in a randomised controlled trial in Primary Care (Anon, 1996). Although education programmes are advocated for TMD, they have not been adequately evaluated. Relaxation training, however, has been shown to be equally effective as conventional occlusal splint therapy Brooke & Stenn, 1983) and benefits may be longer-lasting (Turk et al, 1993). TMD patient improvement after conservative dental treatment was modestly associated with changes in beliefs and coping *with and without* a brief cognitive-behavioural intervention (Turner et al, 1995), suggesting that such changes may accompany simpler treatments. A single trial of a psycho-educational group intervention showed modest but enduring reduction in TMD-related interference compared to usual treatment (Dworkin et al, 1994).

13.III. DEVELOPMENT OF THE CD-ROM

Firstly, a voiceover script was written an rewritten may times over, including information on TMD and self-management advice. This was discussed with focus groups of patients from the previous studies, health psychologists, a clinical psychologist, dentists, maxillofacial surgeons, a psychiatrist, an education

technologist, and a television scriptwriter. It was essential to avoid technical jargon which might not be familiar to patients, whilst limiting the information to the evidence base. A final version evolved (**Appendix VI**), to which illustrative video images were then added.

The programme contains the following sections about jaw pain (TMD): What is it? Who gets it? What causes it? What treatment is available? What can I do? Summary. The programme was designed to provide reassurance as to the benign self-limiting nature of the condition, dispel myths about cause and consequence, reduce pain-related anxiety, and to enhance perceived control and self-efficacy over pain.

Figure 13.I. Illustration of significant factors in TMD, from the preceding studies:

Experience→	*Past pain →*	*Illness perceptions→*	*Coping→*	*Reaction*
Lack of Education	Constant Pain	Consequence Timeline Control/Cure	Catastrophising	Current pain Disability: eating, self-image, oral pain, speech, taste/digestion Mood

The previous studies found disability factors in TMD to include: problems with eating, self-image, oral pain, speech, and taste / digestion (**Figure 13.I.**). These were consequently discussed in the script, with suggestions as to how to minimize the consequence of the condition. In addition, catastrophising was found to be a consistent factor in disability and distress, and a simple attempt was therefore made in the script to discourage such thoughts, by emphasizing how control over pain

might be enhanced – control/cure being a predictor of pain intensity (Chapter 12, p.231), and how symptoms would not be permanent – timeline being another significant factor from previous study (Chapter 12, p.231). The intermittent character of TMD pain was also described, so as to discourage ideas of constant pain, perceived as more consequential (Chapter 11, p.202).

The script was then accompanied by photographic and diagramatic images for illustrative purposes. The full CD for the active programme is attached to the thesis.

13.III.i. **Technical Report**

The voiceover for the CD was edited and manipulated using CoolEdit 95 (Syntrillium Software Corporation, 1995). Original photographs were captured using Nikon CoolPix 950 and Kodak CD 215 digital cameras. Other photographs were sourced from the Photo-Objects Collection (Hemera Technologies Inc., 1998). Images were manipulated and edited in Paintshop Pro version 5 (JASC Software Inc., 1998) and Adobe Photoshop version 5.5 (Adobe Systems Inc., 1999). The elements were brought together using Macromedia Flash version 4 (Macromedia Inc., 1999). The Flash movie settings were 100% image quality and 22MHz mono sound. Given the technology used, the program can, with additional editing, be reconfigured to stream across the internet rather than run from a CD. The resulting CD requires a PC running Windows 95, 98 or 2000 with pointing device, sound card, 64MB RAM and an 8-speed CD player.

13.IV. PROCEDURE

Forty-one TMD patients awaiting consultation appointments were invited to participate in a pilot trial of the programme. Local Ethical Committee approval was obtained for the trial as a continuation of the research already in progress. Patient volunteers were examined to establish the diagnosis according to the research criteria (RDC/TMD, **Appendix IV**) and informed consent obtained to the trial. Patients were then randomly allocated to one of three ten-minute programmes:

Condition 1: An attention placebo CD-ROM comprising anatomical information on the temporomandibular joint and muscles of mastication.

Condition 2: Information on CD-ROM designed to empower patients (see Salmon et al, 1999), increasing control and self-efficacy.

Condition 3: Information on CD-ROM as **2.** with an additional introduction to self-relaxation techniques, followed by an audiotape of progressive muscle relaxation exercises to be despatched from a central source (i.e. independent of the researcher).

13.V. MEASURES

Age, sex and demographic characteristics including marital status, occupation and years in full-time education.

Oral Health Impact Profile (OHIP; pp.30, 136)

Coping Strategies Questionnaire (CSQ; pp. 86, 140)

McGill Pain Questionnaire (SF-MPQ; p.137)

Perceived Stress Scale (PSS-10: p.141)

State-Trait Anxiety Inventory (STAI-6; p.137)

Beck Depression Inventory (BDI; pp. 116, 137)

Self-Efficacy Scale (SES; p.139)

Intervention value scale (IVS)

Multidimensional Health Locus of Control scale (MHLC; p.98)

Health Care Utilisation scale (HCU), for TMD-related consultations as well as analgesic use (self-derived).

Pain Stages of Change Questionnaire (PSOCQ; p.141)

13.VI. RESULTS OF THE PILOT STUDY

13.VI.i. Mean Scores

Table 13.I. shows the scores for the pilot sample. Due to the small size of the pilot sample, inter-group differences were not statistically significant.

Table 13.I. Mean scores (and SD's) or percentages (for dichotomous variables) for the three randomly-selected groups of the pilot sample.

	All	Condition 1	Condition 2	Condition 3
N	41	12	15	14
Age (years)	37 (13)	37 (13)	36 (12)	42 (16)
Sex (% female)	89 %	92 %	93 %	79 %
number of years in Education	15 (4)	13 (5)	17 (4)	15 (3)
Duration (months)	53 (67)	95 (111)	45 (42)	46 (21)
Frequency (days per month)	22 (10)	26 (7)	21 (10)	19 (12)
Deviation on opening (%)	17 %	33 %	20 %	8 %

	All	Condition 1	Condition 2	Condition 3
Opening, assisted with overbite(mm)	38 (7)	40 (6)	38 (8)	36 (7)
Joint sounds (%)	55 %	75 %	53 %	43 %
Myalgia (%)	28 %	25 %	27 %	21 %
Arthralgia (%)	32 %	42 %	20 %	29 %
Disability (OHIP, 0-56)	19 (10)	26 (11)	19 (10)	17 (7)
Past Pain (VAS, 0-100 mm)	57 (21)	63 (16)	59 (25)	46 (21)
Past Pain (VRI, 0-5)	2.5 (1.1)	2.8 (1.0)	2.7 (1.1)	1.8 (1.1)
Anxious mood (STAI, 0-18)	7.3 (4.2)	6.4 (4.6)	7.5 (4.2)	5.9 (2.8)
Depressed mood (BDI, 0-63)	12.5 (11.1)	13.6 (11.5)	11.2 (12.1)	11.4 (9.2)
Health Care visits re. TMD, past 6 months	4 (5)	4 (8)	2 (3)	3 (3)
Analgesic medication (%)	40 %	25 %	40 %	21 %
Prophylactic medication (%)	9 %	17 %	7 %	0
IPQ – Consequence (11-55)	26.9 (7.4)	30.0 (8.9)	24.7 (7.2)	25.2 (4.1)
Permanence (3-15)	8.9 (2.6)	8.6 (1.2)	8.6 (2.9)	8.4 (2.5)
Cyclic (4-20)	12.3 (3.4)	12.6 (2.6)	12.0 (4.3)	12.7 (3.2)
Puzzling (1-5)	3.5 (1.0)	3.4 (.9)	3.1 (.9)	3.7 (1.0)
Cure (5-25)	16.7 (2.4)	16.7 (2.6)	17.1 (2.9)	16.7 (1.0)
Personal Control (7-35)	22.0 (3.6)	22.0 (2.3)	22.1 (3.0)	22.9 93.7)
CSQ - Diverting attention (0-36)	6.1 (5.9)	7.4 (4.3)	5.6 (5.8)	6.1 (7.8)
Increasing behavioural activities (0-36)	12.3 (10.1)	14.0 (10.5)	11.5 (10.8)	11.9 (10.5)

	All	Condition 1	Condition 2	Condition 3
Ignoring sensations (0-36)	6.2 (6.9)	5.4 (5.6)	5.6 (6.9)	7.0 (8.4)
Reinterpreting sensations (0-36)	18.9 (8.5)	19.4 (6.4)	20.0 (7.0)	17.4 (11.8)
Coping self-statements (0-36)	14.4 (8.0)	13.7 (7.8)	13.7 (6.5)	15.6 (10.4)
Praying / Hoping (0-36)	9.6 (7.8)	12.5 (7.9)	8.9 (9.0)	7.1 (5.6)
Catastrophising (0-36)	8.9 (8.7)	10.9 (9.4)	9.9 (9.9)	4.4 (3.8)
Control (0-12)	3.9 (3.5)	4.7 (2.9)	3.7 (4.0)	3.8 (3.5)
MHLC – Internal (6-36)	22.7 (4.9)	22.7 (4.8)	24.6 (5.2)	22.1 (3.0)
External (Chance) (6-36)	17.7 (5.5)	17.9 (5.3)	18.6 (6.5)	16.2 (5.0)
External (Powerful Others) (6-36)	14.4 (6.0)	16.6 (5.2)	12.8 (5.2)	15.4 (7.1)
SES – Self-Efficacy (0-30)	20.1 (5.4)	18.8 (3.5)	20.8 (5.4)	20.6 (4.9)

	All	Condition 1	Condition 2	Condition 3
At T_2 (6 weeks): N	37 (90 %)	10 (83%)	15 (100 %)	12 (86 %)
Disability (OHIP, 0-56)	16.8 (11.0)	21.3 (12.0)	15.4 (12.2)	14.8 (7.7)
Past Pain (VAS, mm)	41 (29)	52 (35)	45 (30)	27 (18)
Past Pain (VRI, 0-5)	1.8 (1.2)	2.3 (1.4)	1.9 (1.2)	1.3 (.6)
Anxious mood (STAI, 0-18)	6.8 (5.0)	7.6 (6.4)	7.1 (5.2)	5.9 (3.4)
Depressed mood (BDI, 0-63)	9.3 (9.9)	10.9 (8.1)	8.5 (12.0)	8.8 (9.3)
PSOCQ – Precontemplation (7-35)	19.7 (6.2)	22.2 (7.0)	18.1 (4.7)	19.4 (6.9)
Contemplation (10-50)	33.0 (6.9)	35.2 (4.0)	33.5 (6.7)	30.6 (8.7)
Action (6-30)	18.3 (5.1)	17.3 (4.6)	18.9 (5.5)	18.6 (5.1)
Maintenance (7-35)	22.4 (5.6)	20.7 (5.1)	23.1 (5.3)	22.9 (6.3)

13.VI.ii. **Correlations**

Correlation analysis was conducted for the entire group. Significant correlations ($p<0.01$) were found for the principal dependent variables.

Anxious mood (STAI) correlated with depressed mood (BDI, 0.645) and catastrophising (CSQ, 0.413). Depressed mood also correlated with disability (OHIP, 0.592) and consequence (IPQ, 0.413). Disability also correlated with: pain (VRI, 0.459); consequence (0.588); diverting attention (CSQ, 0.440); and catastrophising (0.432). Pain over the past month (verbal rating) correlated with: pain (VAS, 0.491);

frequency (0.436); disability (0.459); consequence (0.551); and catastrophising (0.675). Monthly frequency of pain also correlated with belief in the permanence (IPQ, 0.468) and negatively with cyclical nature (IPQ, -0.516) of the pain. Health care visits correlated negatively with a belief in cure (IPQ, -0.439).

13.VI.iii. **Differences in Means**

In addition, there were a few significant differences in means between groupings on dichotomous variables (**Table 13.II.**).

Table 13.II. Significant Differences in Means ($p<0.01$):

Grouping	N	Duration (months)	Consequence (IPQ)	Increasing Behaviour (CSQ)	Coping self-statements (CSQ)	Ignoring sensations (CSQ)
Female	40	56 (71)	-	13.9 (9.8)	-	-
Male	5	23 (13)	-	2.0 (2.6)	-	-
mandibular Deviation	8	-	-	-	-	21.3 (6.0)
none	37	-	-	-	-	12.9 (7.7)
Myalgia	13	-	33.2 (7.3)	-	-	-
none	32	-	24.6 (6.1)	-	-	-
Arthralgia	15	-	-	-	24.4 (6.7)	19.6 (7.6)
none	30	-	-	-	16.3 (8.0)	11.5 (6.9)

13.VII. FEEDBACK

Feedback questionnaires were considered for the entire group together so as not to compromise blinding of the investigator to group. Feedback about the programme was generally positive (Intervention Value Scale), with patients appreciating the discussion of cause and treatment options, and the self-management advice, whilst some expressed understandable disappointment at the lack of a definitive cure. Patients were asked to rate the programme on Likert-type scales of 1 to 5 for 'bad' to 'excellent', resulting in means (and standard deviations, SD) of 4.1 (0.9) for overall usefulness, 4.1 (0.9) for usefulness of information, and 3.8 (1.0) for usefulness of advice. Similar scales were used to rate the programme in relation to a written leaflet (4.2, SD 0.8) and to a personal consultation (3.3, SD 1.1). 78% of patients considered the length of the programme to be appropriate, and 85% felt they were likely to practice any self-help suggestions from the programme.

13.VIII. DISCUSSION

In this, the third study, it was hypothesized that the two experimental conditions would be equally effective in reducing pain, disability and distress, and both more effective than the attention placebo condition. Primary outcome improvements were expected to be associated with modest ameliorations in pain-related cognitions, including locus-of-control and self-efficacy.

Pain control through education and self-management, apparently effective in other conditions (Jensen et al, 1999) might be possible in TMD, through manipulation of the cognitive factors assessed in the above studies. The ultimate goal is an intervention specifically aimed at ameliorating cognitions and behaviours in TMD, in order to reduce the associated pain, disability and distress. A pilot study of a psycho-education self-management programme was conducted in a group of TMD patients. Although not statistically significant, the experimental groups (2 and 3) appeared to have improved at follow-up relative to the placebo group (1) in terms of disability, pain and depressed mood. However, it should be noted that the groups, although randomly selected, did in fact differ at the outset, and these trends must be discounted.

There was some loss to follow-up and this was greatest in the placebo group (Condition 1) and in the relaxation group (Condition 3). This would be a risk in a full study but does not imply that such would be unfeasible. The pilot has demonstrated the feasibility of the study design and acceptability of the approach, particularly in Condition 2 (100% follow-up).

Chapter 14. GENERAL DISCUSSION

Much research has been conducted on psychological aspects of chronic pain syndromes, yet, as a domain within dentistry, orofacial pain has tended to be seen as a special case and managed by orofacial specialists. This thesis has attempted to bring the understanding of orofacial pain, and of temporomandibular disorders (TMD) in particular, into line with that of other chronic pain conditions.

The three studies reported above have investigated cognitive factors, which relate to perceived control, disability and distress in TMD patients.

14.I. INTER-GROUP COMPARISON

TMD groups were compared with an acute pain group of known cause and timeline (post-extraction pain) and with another idiopathic chronic pain group, in which psychological factors are accepted as contributory (primary headache: De Benedittis & Lorenzetti, 1992; Spierings et al, 1997). Comparison with post-extraction pain in terms of disability aimed to discriminate thus between the two pain groups, one knowing the cause and timeline of their pain, the other not. Comparison with the headache group aimed to determine the contribution of pain location and character to cognitions and outcome, in two idiopathic pain groups.

14.I.i. TMD and Post-Extraction Groups

In the first study, a group of patients with a history of painful TMD (chronic pain model) was compared with a group of patients who, three days previously, had had wisdom teeth surgically removed (acute pain model). The Oral Health Impact Profile

(OHIP) was used to measure physical, social and psychological disability. There were significant differences between the two groups on two factors: the post-extraction group was more functionally limited and more physically disabled than was the TMD group ($p<.01$). These two factors comprise the *physical* aspects of disability, and, hence, the differences illustrate the greater *physical* impairment associated with the acute condition, which may last days, in comparison with the chronic condition, which may last years. There were no significant inter-group differences on the social and psychological subscales of the OHIP, which might have been expected since TMD has previously been associated with psychological distress (Gatchel et al, 1996). The implication from the present study is that wisdom tooth removal is comparably distressing.

Post-operative pain appeared to have a more obviously physical component to it than did TMD, and yet two earlier studies of post-extraction pain found anxiety pre-operatively predicts pain intensity post-operatively (George et al, 1980; Feinmann et al, 1987). In the light of this psychological predictor of acute pain, and target for preventative manipulation, the possibility of psychological targets in TMD, a condition associated with significantly less physical disability, presents itself for investigation.

14.I.ii. **TMD and Headache Groups**

14.I.ii.a. ***Duration and Pain Intensity***

In the second study, headache patients had endured their symptoms for longer than had the TMD group, and reported greater pain intensity ($p<.01$) at the time of their initial hospital consultation. Patients were also asked to describe the character of

their pain using the McGill Pain Questionnaire (MPQ). The headache group's description of pain differed from that of the TMD group, but, in reporting *both* greater "constant" pain *and* greater "intermittent" pain, this would appear to be more a reflection of greater overall pain intensity than any distinction in character.

This failure of the MPQ to discriminate between the two pain conditions may relate to the heterogeneity of the groups, with TMD including muscle and joint problems and headache including tension-type and migraine. Other authors have found the MPQ to distinguish, for example, between myogenous and arthrogenous TMD (Mongini & Italiano, 2001).

The inter-group differences in pain intensity and duration might suggest that individuals will tolerate headaches more than they will TMD, before seeking help from secondary healthcare; or, alternatively, that general medical practitioners are more reluctant to refer to secondary care than are general dental practitioners (the prime source of referral for TMD). Both these interpretations may reflect the greater prevalence of headache than of TMD (up to 30% vs. 6% repectively; Rasmussen & Olesen, 1994; Lipton et al, 1993); and therefore, perhaps, a greater familiarity with headache and lesser threat from it, in the general population. Thus, it is only when headache sufferers have endured their symptoms for some considerable time and at a considerable intensity that they are referred to hospital; and, conversely, TMD sufferers are referred earlier. The prevention of chronic recalcitrant problems by early intervention might therefore be aided in TMD by this readier referral pattern.

14.I.ii.b. ***Illness Perceptions, Coping Strategies and Alexithymia***

The headache sufferers considered their symptoms to be more consequential and less controlled than did the TMD sufferers ($p<.01$: Illness Perceptions Questionnaire, IPQ; Coping Strategies Questionnaire, CSQ), even after allowing for differences in pain duration and intensity.

Headache patients also catastrophised ("It's terrible and I feel it's never going to get better" etc.) more than did the TMD group ($p<.01$: CSQ). Statistical analysis of covariance, however, found this difference in catastrophising to be explained by the greater duration and pain intensity of the headache group. Thus catastrophising increases the longer the pain continues. This finding supports the suggestion of previous researchers in describing a cognitive shift in headache sufferers away from situational and interpersonal distress and towards distress associated with the disorder itself, i.e. catastrophising (Demjen et al, 1990).

This cognitive shift is presently also reflected in two other factors: "difficulty describing feelings" and "externally-oriented thinking", measured by the Toronto Alexithymia Scale (TAS). Again the headache group scored higher than did the TMD group on both of these factors ($p<.01$). Although alexithymia (literally "no words for mood") has been conceived as "a unique constellation of personality traits that may predispose to chronic pain and other disorders" (Lumley et al, 1997, p.163), this is not supported by other personality measures, which show no such predisposition in pain groups (Wade et al, 1992). An alternative conception might be that alexithymia is a coping style, which may be adopted in response to the ongoing presence of a disabling and distressing condition such as chronic pain.

14.I.ii.c. ***Distress and Medication***

One aspect of the longitudinal data from the second study is the remarkable consistency of factors, including pain beliefs, coping strategies, disability and distress. Beliefs and coping, in particular, are conceived by cognitive-behavioural therapists as labile cognitions, influencing and being influenced by changing stressors and stress. However, in the present second study, apart from pain and distress in the headache group, very little appears to have changed over six months. This is in considerable contrast to previous evidence of cognitive and behavioural improvement with treatment, even if that treatment was not cognitive-behavioural in approach (Turner et al, 1995). The TMD group studied here quite simply failed to improve despite conventional management, which included reassurance, splint therapy and anti-depressant medication.

The headache group, reporting initially greater pain intensity and depressed mood, showed significant improvement in these measures of outcome, both absolutely and relatively to the TMD group ($p<.01$; Figure 12.II, p.213.). The only measured correlate that might help to explain this inter-group difference in outcome is that of prophylactic medication (principally with tricyclic anti-depressants), the prescription of which was greater in the headache group than in the TMD group, and was associated with greater improvement in distress ($p<.01$; Figure 12.III, p.215). A randomised controlled trial of tricyclic anti-depressant treatment in headache has indeed found it to be more effective than placebo in reducing headache activity and headache-related disability (Holroyd et al, 2001).

It may well be that more widespread prescription of prophylactic medication for the TMD group would have had a beneficial effect, as seen in previous studies (Feinmann & Harris, 1984,ii; Tversky et al, 1991), or that non-pharmaceutical means may have reduced anxiety (Turner et al, 1995; Turk et al, 1996; Harrison et al, 1997).

14.II. INFLUENCE OF COPING STRATEGIES ON OUTCOME

A review of the literature on coping in chronic pain patients demonstrated consistent evidence of the association of adaptive coping strategies (e.g. reinterpreting pain sensations, coping self statements) with better adjustment, and of maladaptive strategies (catastrophising, praying/hoping) with worse adjustment, despite methodological inconsistencies. Variation across pain groups had been noted and warranted further investigation before general conclusions could be drawn regarding coping in chronic pain. The relationship between changing coping strategies and development of chronicity of pain symptoms was considered likely to provide useful insight into the aetiology of chronic pain.

14.II.i. Coping Strategies and Pain Intensity

In the study reported above (Chapter 12), coping strategies endorsed initially did not contribute to the report of current pain intensity at 6 months.

Previous studies have found relationships between coping and pain. Using the Ways of Coping Scale in TMD patients, Schnurr et al (1991) found coping strategies to explain 51% of the variation in pain intensity. Using the Coping Strategies Questionnaire, Hill et al (1995) found catastrophising to be associated with greater phantom pain intensity in amputees; and Nicassio et al (1995) found high active

coping (diverting attention, reinterpreting sensations, coping self-statements, increasing behavioural activity) and low perceived control over pain to predict greater pain intensity in a longitudinal study of fibromyalgia patients. The TMD study (Schnurr et al, 1991) used the WCS, which has no specified stressor, as opposed to the CSQ, which specifies pain as the stressor; this may have introduced confounding elements such as psychosocial dysfunction into the relationship with pain.

The lack of prediction of pain over time may be partially explained by the intervening effect of illness perceptions, as seen in a study of cancer patients (Barkwell, 1990). In the present study, change (decrease) in pain intensity over 6 months did correlate significantly and positively with a corresponding change in catastrophising (.472, $p<.01$), suggestive of a relationship, albeit one which did not contribute to a prospective prediction of pain, again possibly because of intervening illness perceptions, such as consequence and control/cure. This confirms a previous longitudinal study of multidisciplinary pain treatment, in which decrease in catastrophising was associated with decrease in pain intensity (Jensen et al, 2001).

14.II.ii. Coping Strategies and Disability

Turner et al (2000) found active coping scores to predict physical disability in a longitudinal study of pain clinic patients. No relationship between active / adaptive coping strategies and disability was found in the present study, although, again, change in catastrophising over 6 months correlated significantly and positively with a corresponding reduction in disability (.367, $p<.01$). A more recent study by Turner and colleagues (2001) also failed to replicate an independent association of coping

with disability, confirming that this is not a consistent finding, and that the influence of coping strategies on disability may be affected by other factors, such as illness perceptions.

14.II.iii. Coping Strategies and Psychological Distress

Catastrophising explained 14% and 34% of the variance in anxious and depressed moods, respectively, in the first study's TMD group, confirming the relationship between catastrophising and distress found previously (Dozois et al, 1995; Turner et al, 2000). In the second study, initial catastrophising failed to predict later distress longitudinally, although there was a significant positive correlation between change in catastrophising and change in anxious mood (.391, $p<.01$). There were also no significant relationships between active coping strategies and distress. This may well reflect a weakness in the CSQ as, unlike other coping measures (WCCL, VPMI), active coping factors do not relate to distress, as noted earlier in the literature review.

14.III. INFLUENCE OF ILLNESS PERCEPTIONS

The conceptually related ideas of locus of control, pain (i.e. causal) beliefs, self-efficacy and expectancy have associations with coping and adjustment in chronic pain. In addition, pain beliefs are associated with physical disability and depression, independently of coping and catsatrophising (Turner et al, 2000). With regard to temporomandibular pain, given its unknown aetiology, patients' beliefs were considered of particular interest.

14.III.i. Causal Attributions

14.III.i.a *Psychological Cause*

In the first part of the second study (Chapter 11), an attempt was made to explain how causal attributions might arise. Attribution of pain to a psychological cause (stress, own behaviour, state of mind) was partly explained by shorter time in education (6%) and lesser frequency of pain episodes (12%). The relation with education may reflect socio-economic factors omitted from this study, or a greater emphasis on 'hard' scientific explanation for pain in those who had spent longer in education.

How causal attributions might then influence disability and distress, was examined in both first and second studies using two different instruments: the pain Beliefs Questionnaire (pp.100, 138) and the Illness Perceptions Questionnaire (pp.101, 139). In the first study (Chapter 10), psychological pain beliefs contributed to 1% of the variance in anxious mood, and, in the second study (Chapter 12), initial attribution to psychological cause correlated with distress at 6 months, although contribution to the regression equations for anxious and depressed moods was insignificant. These associations, though weak, are similar to those found in chronic fatigue syndrome sufferers (Moss-Morris et al, 1996), in whom a belief in stress as causal was associated with greater disability and distress.

Previous comparison of the Pain Beliefs and Perceptions Index (PBPI) and the Multidimensional Health Locus of Control scale (MHLC) has found belief in pain as *mysterious* to relate to internal locus of control, and to psychological distress (Williams & Thorn, 1989; Herda et al, 1994; Williams et al, 1994).

14.III.i.b. ***Physiological Cause***

Attribution to physiological cause (germ / virus, diet, pollution, heredity) in the second study was influenced by lower frequency of pain episodes as well as by 'constant' pain character, demonstrating a clear perceived relationship between pain characteristics and causal beliefs. As to how this causal attribution might in turn be of influence, neither study reported here found any relationship between physiological causal beliefs and disability / distress. Interestingly, in the second study, the headache group reported significantly greater belief in physiological cause than did the TMD group, at 6 months (8.9, SD 3.3 vs. 6.9, SD 2.7; range 4 – 20; $p<.01$); this inter-group difference was less significant initially. It may be that a greater belief in physiological cause in the headache group contributed to greater improvements in pain and distress.

14.III.i.c. ***External Cause***

Attribution to external cause (chance, other people, poor dental care) was partly explained by lesser pain intensity (10%) and non-intermittent character (1%); and had no relationship with disability / distress. This contrasts with a previous finding of an association between 'chance' locus of control and distress (Crisson & Keefe, 1988). Previous comparison of the MHLC and Pain Beliefs Questionnaire (PBQ) found 'chance' and 'powerful others' locus of control subscales to relate to 'organic' beliefs (Edwards et al, 1992). In the first study reported here, organic beliefs on the PBQ were unrelated to disability / distress. This suggests that locus of control may be more influential on distress than are beliefs measured by the PBQ.

14.III.ii. **Timeline**

Variance in perceived timeline (i.e. permanence of pain: "My pain will last a long time", "My pain is likely to be permanent rather than temporary") was explained in the second study by shorter education (19%) and greater pain intensity (1%), together with a lack of belief in controlability / curability (24%).

Change in perception of timeline over time in the second study correlated significantly and positively with changes in disability and anxious mood (.372 and .509 respectively, $p<.01$), yet initial perception of timeline did not significantly predict final disability (–1%). This is in line with previous comparison of the PBPI and the MHLC, in which belief in pain as *enduring* related to internal locus of control, and to psychological distress (Williams & Thorn, 1989; Herda et al, 1994; Williams et al, 1994). Belief in one's pain as long-term or permanent is therefore a maladaptive belief.

14.III.iii. **Consequence**

Variance in perceived consequence of pain was explained by shorter education (9%), greater duration of pain at time of consultation (6%), greater intensity of pain (13%), 'constant' pain character (12%), location of pain (TMD or headache: 2%), physiological cause attribution and perceived timeline (5%). A total 47% of the variance was explained in the regression equation, which suggests that the factors considered are appropriate to the investigation, being significant contributors to the explanation of perception of consequence.

In the longitudinal analysis, initial perception of consequence was itself an insignificant contributor to the variance in pain intensity at six months (5%, together

with Control/Cure), and to the variance in depressed mood at six months (2%, together with psychological cause); yet change in perceived consequence over that time correlated significantly and positively with change in disability (.365, $p<.01$), and, less significantly, with changes in pain intensity and anxious mood (.290 and .284 respectively, $p<.05$). Change in perceived consequence also correlated with change in catastrophising (.330, $p<.01$) and, negatively, with changes in coping self-statements (an active strategy) and perceived control (CSQ: -.329 and -.420 respectively, $p<.01$).

Perceived consequence would therefore appear to be a useful indicator of many dimensions of the pain experience. Comparable results were obtained by Moss-Morris et al (1996) for Chronic Fatigue Syndrome (CFS) sufferers: those believing their illness to be of serious consequence wer most disabled and distressed. Similarly, in another study of TMD patients, using the Survey of Pain Attitudes, those believing their pain to be disabling were more disabled and depressed (Turner et al, 2001).

14.III.iv. **Control / Cure**

The final factor of the Illness Perceptions Questionnaire, control / cure, was poorly explained by the other factors considered, with only the other cognitions, timeline (negatively) and psychological cause, contributing with any magnitude to the variance (20%). In turn, longitudinally, control / cure explained 5% of the variance in pain intensity (negatively), and change in perceived control / cure correlated significantly, and negatively, with changes in perceived timeline and consequence (-.424 and -.410

respectively, $p<.01$). Change in control / cure (IPQ) also correlated significantly with the perceived control factor of the CSQ (.353, $p<.01$), demonstrating cross-validity. The prediction of pain intensity over six months is highly significant and comes after controlling for initial pain intensity. Previous research has found perceived control over pain to be associated with better psychological functioning, mediated by coping strategies, and with greater physical activity (low pain only), even after controlling for coping, in a mixed chronic pain group (Jensen & Karoly, 1991). Moss-Morris et al (1996) also found the belief that the illness is out of control is associated with greater disability and distress in CFS patients.

Self-efficacy over pain is a related concept to control / cure, and has been shown to relate to pain reduction and improvement after treatment (Kores et al, 1990; Buckelew et al, 1994; Anderson et al, 1995). Self-efficacy has also been found to determine treatment-seeking in fibromyalgia sufferers (Kersh et al, 2001).

14.IV. DISABILITY

Previous research into the disability of TMD has found similarities to other chronic pain conditions (Pain Disability Index; Bush & Harkins, 1995). Murray et al (1996) used the Oral Health Impact Profile (OHIP, p.30), an orally specific instrument developed from the widely used Sickness Impact Profile, to assess disability in a craniofacial pain group. In the first study described above, scores on the OHIP were compared with the subgroups from the Murray study, and similar levels of physical, psychological and social disability were found.

In the first study, OHIP subscales correlated significantly with pain intensity, as determined by the McGill Pain Questionnaire (MPQ), except for the functional and physical subscales, whereas clinical signs (joint sounds, jaw opening, muscle pain on palpation) did not relate to the MPQ scales. Interestingly, those functional and physical subscales were the distinguishing factors on comparison with the acute pain group. Arthralgia (joint pain on palpation) was the only clinical sign to relate to the self-report pain intensity scales. Previous research has found a lack of relationship between changes in clinical signs and improvement in TMD (Ohrbach & Dworkin, 1998), and it may well be that disability measures, such as the OHIP, provide better diagnostic and prognostic indicators.

The principal component analysis of the TMD group's responses on the OHIP derived six components: mood impact, problems with eating, self-image impact, oral pain, problems with speech, and disturbance in taste and digestion. Previous studies have reported functional problems such as reduced chewing ability (Kurita et al, 2001) and discomfort in relation to chewing (for example: Reisine et al, 1989), but

not such aesthetic aspects of disability as taste disturbance. It might be that taste sensation is confused with pain, or more likely that this reported impairment is a reflection of the loss of pleasure derived from eating. This is an important issue for any management programme to address, and even a reason in itself for treatment-seeking.

14.V. DISTRESS

14.V.i. Anxious Mood

In the first study, indviduals with higher levels of anxious mood were found to have more pain, confirming previous research (McCracken et al, 1993; Casten et al, 1995). Interestingly, subjects with stronger beliefs in a psychological influence on their pain tended to have higher levels of anxious mood. This is similar to findings with CFS sufferers, as mentioned above (Moss-Morris et al, 1996), and one explanation might be that a heightened state awareness of psychological stress increases the endorsement of such stress as potentially causal of pain. In other words, individuals feeling stressed are more likely to cite stress as causing their pain symptoms, which, in turn, increases anxiety. The influence of "daily hassles" on pain frequency and intensity has previously been established in headache (Fernandez & Sheffield, 1996), and the present second study found both perceived stress and attribution of pain to psychological cause to correlate with levels of anxious mood six months later, even though their predictive power was insignificant after controlling for initial anxious mood (1% and 0% respectively).

Despite methodological problems in previous rsearch, there is evidence of greater prevalence of anxious symptoms in chronic pain populations than in controls (eg.

Krishnan et al, 1985). However, there is a lack of prospective studies to elucidate the causal direction of the anxiety / pain relationship. Since there is compelling evidence of normal personality structure in chronic pain patients (eg. Wade et al, 1992; Schnurr et al, 1990), it seems likely that anxiety develops as a sequela to pain but then contributes to its endurance and impact.

14.V.ii. **Depressed Mood**

In the first study, depressed mood was largely explained by passive coping, and catastrophising in particular, which is consistent with research on other chronic pain groups (for example: Snow-Turek et al, 1996). Disability, disturbance in taste and digestion in particular, was also contributory.

It should be noted that the TMD patients studied here reported only modest levels of depressed mood (study 1: mean 4.8, SD 3.9; study 2 at T_1: 4.1, 3.9; HAD scale range 0 – 21), suggesting that depression is not a prevalent complication of TMD.

Recent evidence suggests depression to be a sequela of pain, or even a sequela of disability (Pincus & Williams, 1999). A "diathesis-stress" framework has been proposed (Banks & Kerns, 1996) to explain the high comorbidity between chronic pain and depression. This approach encourages identification of vulnerability factors in the individual as well as investigation into the nature of the stressor, and provides a useful theoretical basis from which to advance the study of depression in chronic pain.

14.V.iii. **Distress and Chronicity**

The concept of anxiety as a positive strategy for acute pain, in that expected sudden pain is perceived as likely to be less intense than unexpected pain (Madland, 1988), may well be analagous to the prevalence of anxiety in acute TMD populations relative to that in chronic populations (Gatchel et al, 1996). Anxiety may contribute to the interpretation of peripheral sensations as painful early on in the course of the condition, and then be superceded by depression when its lack of effect on the persistence of pain is realised, in those individuals who fail to respond to current therapies, due to inappropriate beliefs regarding the cause and consequence of their pain, maladaptive coping strategies, and lack of perceived control. Although previous investigation has shown anxiety to be a feature in acute TMD patients and depression a feature in chronic TMD patients (Gatchel et al, 1996), this was not confirmed in the present mixed-duration sample (mean duration 30 months: first study), where there were no relationships between pain duration and anxiety or depression. This difference may be due to the relative sensitivities of the different diagnostic instruments used.

14.VI. PATIENT EDUCATION AND SELF-MANAGEMENT

Although assessed in the present studies by no more than a simple question of time spent in full-time education, a lack of education contributed significantly to attribution of pain to a psychological cause, and to perceptions of timeline and consequence. All three of these cognitive factors were in turn related to disability and / or distress. A logical extrapolation of these findings would be the incorporation of an education programme for sufferers of TMD into a management strategy.

Another aspect of a self-management strategy is improvement in self-efficacy, which, being related to perceived control / cure, ought to translate into a reduction in pain over time, control / cure being predictive of pain intensity.

In the third study, the differences in perceptions and coping strategies seen between patients with or without pain on palpation of the temporomandibular joint (arthralgia) or muscles of mastication (myalgia) suggest two distinct disorders. Patients perceiving their pain as of great consequence were more likely to report myalgia, whilst patients utilising the supposedly positive strategies of coping self-statements and ignoring sensations were more likely to report arthralgia. This is in line with Epker et al's claim (1999: see Chapter 2, p.33) that *myalgic* patients are more likely to develop chronic symptoms of TMD; and with Auerbach et al's findings (2001) that psychological factors play a greater role in myalgia than in arthralgia.

14.VII. SHORTCOMINGS OF THE STUDIES

14.VII.i. Recruitment, Retention and Compliance

Serious failings are evident in the studies, particularly for the comparison groups. This illustrates the necessity for the principal investigator to give equal attention to comparison groups, even, and especially, where those participants are unlikely to benefit in the long-term from the research in hand, which may be perfectly transparent. The purpose of the research may not have been adequately explained to comparison groups.

Recruitment of the third molar patients in the first study was left to outpatient clinic staff, whose help is gratefully acknowledged but whose incentive in impressing the need for compliance upon potential participants may not have equaled that of the principal investigator. Third molar patients were simply given a stamped addressed envelope in which to return their questionnaire on completion three days post-operatively. Consequently, compliance was poor with only 50% of questionnaires being returned, compared to excellent compliance in the TMD group, who were all personally recruited and examined by the principal investigator. Although post-operative review of third molar patients is not routine, such an addition, in combination with the recovery of questionnaires, would presumably have greatly improved compliance.

In the second study, attrition rates were high for the longitudinal assessment: 25% in the TMD group and 35% in the headache group. Whilst this may be explained by the lack of incentive to comply in the absence of any treatment on offer, improved communication and correspondence might well have improved retention of subjects.

The drop out and self-selection of the other groups mean that results can only be viewed as comparative, and not as representative of third molar or headache patient groups *per se*.

14.VII.ii. **Confounding Group Differences**

The younger age of the post-extraction group in the first study, and the greater pain duration and intensity of the headache group in the second, made these less than ideal groups for comparison, despite controlling for these differences wherever possible in the analyses.

14.VII.iii. **Self-Report Questionnaires**

The reliance on self-report measures risks different scales effectively measuring the same construct, for example catastrophising and depressed mood. The poor correlation with observable clinical signs, however, illustrates the difficulty in finding objective measures in TMD.

Selecting the most informative and appropriate instruments is often a matter of trial and error. The Pain Beliefs Questionnaire (PBQ) appeared to be ideally suited to TMD but failed to provide factors of any great influence in the first study, and was subsequently replaced with the Illness Perceptions Questionnaire (IPQ) and the Multidimensional Health Locus of Control scale (MHLC). The Hospital Anxiety and Depression scale (HAD), although apparently controlling for confounding of pain and distress, failed to distinguish between an acute pain condition and a chronic pain condition in the first study, which the more thoroughly established State-Trait Anxiety Inventory (STAI) and Beck Depression Inventory (BDI) might have done.

14.VII.iv. **Weighting of assessment in favour of TMD**

TMD being the principal focus of interest, the disability measure and clinical examination in the second study were tailored to TMD rather than to headache, compromising comparison on these aspects. Causal items on the IPQ were similarly biased and factors relating to cancer were not included, despite being a cause of concern to headache patients (Edmeads, 1998). For these reasons, the results for the headache group should be considered as comparative and interpreted with caution. Attrition was also greater in the headache group than in the TMD group, sounding a further note of caution.

14.VII.v. **Lack of change in TMD group**

The minimal changes over time across the board of assessment compromise the prediction of outcome and suggest that further investigation ought to accompany an intervention aimed at enhancing perceived control over pain and reducing pain-related disability and distress, in the expectation of greater changes and better outcome.

14.VII.vi. **Pilot Study**

The pre-existing inter-group differences in the pilot study, in spite of randomisation, negate any observable trends at follow-up. However, the apparent improvement in the placebo group, in terms of disability, past pain, and depressed mood, might suggest that the attention placebo programme wasn't as innocuous as intended. In including anatomical information about the temporomandibular joint and associated

musculature, this programme might have paradoxically been helpfully informative in itself. Changes to the placebo might be warranted in a full-scale trial.

A pilot study derives no statistically significant results and merely demonstrates the feasibility and acceptability of the design. A full, randomized, controlled trial is required to confirm the efficacy of the interventions developed here.

Chapter 15. CONCLUSIONS

15.I. In the first study, the weak association of self-reported disability and clinical signs was confirmed, strengthening the support for a biopsychosocial rather than medical model. The study also demonstrated that, in TMD patients, anxiety is associated with pain, catastrophising the pain, and with 'psychological' pain beliefs. The latter are beliefs that pain is affected (increased) by anxiety and depression. The more anxious patients also emphasise the impact of their illness on their mood, thus effectively endorsing a relationship between anxiety and TMD. In addition, anxiety appears to be related to perceived problems with speech. Depressive symptoms are associated with 'passive' coping strategies, notably catastrophising, and with emphasis on the impact on tasting and digesting food.

15.II. Many researchers and clinicians now advocate cognitive-behavioural approaches to the management of medically unexplained conditions such as TMD and headache, based on the finding that certain cognitions and behaviours are better associated with adaptation to chronic symptoms than are others, and that these should therefore be preferentially encouraged. The second study found that greater intensity of 'constant' pain contributed to greater advocation of physiological cause, and greater perceived consequence of pain on psychosocial functioning, whilst a longer period in full-time education appeared to increase endorsement of psychological causes, and to beneficially influence judgments of timeline and consequence. One conclusion from these latter findings might be a positive therapeutic benefit of patient-education programmes in the conditions here

investigated, as has been seen in other chronic pain conditions such as rheumatoid arthritis.

15.III. TMD, though reportedly less painful, of lesser consequence, and more controllable than headache, was also less responsive to treatment, as demonstrated over six months following hospital specialist consultation. The contribution of perceived permanence and consequence, and of catastrophising, to continuing pain, disability, and distress indicates that these factors are suitable targets for a psycho-educational approach aimed specifically at ameliorating pain beliefs and coping strategies.

15.IV. An evidence-based patient education programme for TMD has been developed on CD-ROM. The pilot study demonstrated the acceptability of the self-management programme and format, as well as the feasibility of conducting a full-scale trial of the programme according to the procedure described.

Key to Abbreviations of Measurement Instruments

AD	Anxiety Differential (Husek and Alexander,1963)
ADS	Arthritis Disability Scale
APQ	Autonomic Perceptions Questionnaire (Borkovec and O'Brien, 1977)
ASI	Anxiety Sensitivity Index (Peterson and Reiss, 1992)
ASQ	Attributional Style Questionnaire
AUDIT	Alcohol Use Disorders Identification Test (Babor and Grant, 1989)
BAI	Beck Anxiety Inventory
BDI	Beck Depression Inventory (Beck et al, 1961)
BHS	Beck Hopelessness Scale (Beck and Weissman, 1974)
BMSS	Broadhead Measure of Social Support
BPPA	Body Parts Problem Assessment scale
BPRS	Behavior Profile Rating Scale (Melamed, 1975)
CCSI	Cognitive Coping Strategy Inventory
CEQ	Cognitive Errors Questionnaire
CES-D	Center for Epidemiology Studies Depression scale
CISS	Coping Inventory for Stressful Situations - adolescent form (Endler and Parker,1990)
CPCI	Chronic Pain Coping Inventory (Jensen et al, 1995)
CPSS	Chronic Pain Self-Efficacy Scale (Anderson et al, 1995)
CSAQ	Cognitive Somatic Anxiety Questionnaire
CSQ	Coping Strategies Questionnaire (Rosenstiel and Keefe, 1983)
DAS	Dental Anxiety Scale (Corah, 1969)
DAS	Dysfunctional Attitude Scale
DAST	Drug Abuse Screening Test (Skinner, 1982)
DBS	Dental Beliefs Survey (Milgrom et al, 1985)
DDS	Descriptor Differential Scale (Gracely et al, 1978)
DFS	Dental Fear Survey (Kleinknecht et al, 1973)
DORS	Dental Operatory Rating Scale
DSM-III-R	Diagnostic and Statistical Manual 3rd edition, Revised
DSM-IV	4th edition (American Psychiatric Association, 1987, 1994)
EPI	Eysenck Personality Inventory (Eysenck and Eysenck, 1964,1968)
EPQ	Ethnic Pain Questionnaire
EPQ	Eysenck Personality Questionnaire (Eysenck and Eysenck, 1975)
FSS	Fear Survey Schedule (Geer, 1965)
GCS	Group Comparison Scale (after Bandura, 1977)
GHQ	General Health Questionnaire (Goldberg, 1972)
GSI	Global Severity Index
HAD	Hospital Anxiety and Depression scale (Zigmond and Snaith, 1983)

HAQ	Health Assessment Questionnaire
HI	Headache Index
HLC	Health Locus of Control scale (Wallston et al, 1976)
HSCL-21	Hopkins Symptom Checklist 21 (Derogatis et al, 1974)
IBES	Illness Behavior Encouragement Scale (Walker and Zeman, 1992)
IBQ	Illness Behavior Questionnaire (Pilowsky and Spence, 1976)
IBQ	Illness Beliefs Questionnaire
IDCI	Iowa Dental Control Index (Logan et al, 1991)
IPQ	Illness Perceptions Questionnaire (Weinman et al, 1996)
LAIS	Life Activity Impact Scale (George et al, 1980)
LLOC	Levenson's Locus Of Control scale
MAPPS	Movement And Pain Prediction Scale
MCMI-II	Millon Clinical Multiaxial Inventory-II
MHLC	Multidimensional Health Locus of Control scale (Wallston et al, 1978)
MIQ	Meaning of Illness Questionnaire (McAdams et al, 1989)
MMPI	Minnesota Multiphasic Personality Inventory (Graham, 1990)
(WHY)MPI	(West Haven - Yale) Multidimensional Pain Inventory (Kerns et al, 1985)
MPQ-SF/PRI(T)	McGill Pain Questionnaire - Short Form or (Total) Pain Rating Index (Melzack, 1987)
MSPQ	Modified Somatic Perception Questionnaire (Main, 1983)
NEO-PI	NEO-Personality Inventory (Costa and McCrae, 1985)
PAI	periodontal Pain Areas Index
PAIRS	Pain And Impairment Relationship Scale
PAIS-SR	Psychological Adjustment to Illness Scale - Self-Report
PANAS	Positive And Negative Affect Schedule (Watson et al, 1988)
PASS	Pain Anxiety Symptoms Scale (McCracken et al, 1992)
PB(A)PI	Pain Beliefs and Perceptions Inventory
PBQ	Pain Beliefs Questionnaire (Edwards et al, 1992)
PCQ	Pain Cognitions Questionnaire
PCS	Pain Catastrophizing Scale (Sullivan et al, 1995)
PDI	Pain Disability Index (Pollard, 1984)
PDQ	Pilowsky's Depression Questionnaire
PDS	Perceived Disability Scale
16-PFQ	16 Personality Factor Questionnaire (Cattell and Eber, 1964)
PLOC	Pain Locus Of Control scale
P(O)MS	Profile of Mood States (McNair et al, 1971)
PRSSS	Pain-Related Self-Statements Scale (German)
PSEQ	Pain Self-Efficacy Questionnaire
PSI	Palmar Sweat Index
PSPI	PsychoSocial Pain Inventory

RCD-D	Research Diagnostic Criteria for Depression
RDC/TMD	Research Diagnostic Criteria for Temporomandibular Dysfunction (Dworkin and LeResche, 1992)
RGWAS	Rand General Well-being Adjustment Scale (Brook et al, 1979)
RLOC	Rotter's Locus Of Control scale (1966)
RSES	Rosenberg Self-Esteem Scale
RSS	Repression Sensitization Scale (Byrne, 1961)
SCID	Structured Clinical Interview for DSM
SCL-90-R	Symptoms CheckList, 1990 (Revised)
SDS	Social Desirability Scale (Crowne and Marlowe, 1960)
SHI	Sternbach Health Index
SHSS	Stanford Hypnotic Susceptibility Scale (Weitzenhoffer and Hilgard, 1959)
SIP	Sickness Impact Profile (Bergner et al, 1981)
SLR	Straight Leg Raising test
SOPA	Survey Of Pain Attitudes (Jensen et al, 1994)
SPIES	Stanford Preschool Internal-External Scale (Mischel et al, 1974)
SPR	Sternbach Pain Rating scale
SPS	Self-Perception Scale
SRE	Schedule of Recent Experience
STAI	Spielberger State-Trait Anxiety Inventory (Spielberger et al, 1970)
STAIC	STAI for Children (Spielberger et al, 1973)
STAXI	State-Trait Anger Expression Inventory (Spielbereger et al, 1985)
SWLS	Satisfaction With Life Scale
TAS	Tellegen's Absorption Scale (Tellegen and Atkinson, 1974)
TAQ	Tellegen Absorption Questionnaire (Tellegen, 1976)
TENS	transcutaneous electrical nerve stimulation
TL	Trouble List (German)
TMAI	Taylor's Manifest Anxiety Inventory
UHI	Ursin Health Inventory (Ursin et al, 1988)
VAS	Visual Analogue Scale
VPT	Venham Picture Test (Bengston and Cipes, 1977)
WCDI	Waddell's Chronic Disability Index
WCQ	Ways of Coping Questionnaire
WES	Work Environment Scale
WPII	Waddell Physical Impact Index
WROS	Waiting Room Observation Scale
ZSRDS	Zung Self-Rating Depression Scale (Zung, 1965)

REFERENCES

Abraham J, Pierce C, Rinchuse D & Zullo T (1992). Assessment of buccal separators in the relief of bruxist activity association with myofascial pain-dysfunction. Angle Orthodont 62: 177-183.

Adair SM & Hecht C (1993). Association of generalised joint hypermobility with history, signs, and symptoms of temporomandibular joint dysfunction in children. Pediatr Dent 15: 323-326.

Affleck G, Tennen H, Pfeiffer C, Fifield J & Rowe J (1987). Downward comparison and coping with serious medical problems. Am. J. Orthopsychiat. 57:570-578.

Aghabeigi B, Feinmann C, Glover V, Goodwin B, Hannah P, Harris M, Sandler M & Wasil M (1993). Tyramine conjugation deficit in patients with chronic idiopathic temporomandibular joint and orofacial pain. Pain 54:159-163.

Aghabeigi B, Feinmann C and Harris M (1992). Prevalence of post-traumatic stress disorder in patients with chronic idiopathic facial pain. Br. J. Oral Maxillofac. Surg. 30:360-364.

Ahles TA, Cassens HL and Stallings RB (1987). Private body consciousness, anxiety, and the perception of pain. J. Behav. Ther. Exp. Psychiat. 18:215-222.

American Psychiatric Association (1987). Diagnostic and statistical manual of mental disorders (3rd ed., rev.). Washington, DC: APA.

American Psychiatric Association (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: APA.

Anderson K, Noel Dowds B, Pelletz R, Edwards WT & Peeters-Asdourian C (1995). Development and initial validation of a scale to measure self-efficacy beliefs in patients with chronic pain. Pain 63:77-84.

Anderson R, Baron R and Logan H (1991). Distraction, control and dental stress. J. App. Soc. Psychol. 21:156-171.

Anonymous (1996). The Back Book. The Stationery Office, London.

Antczak-Bouckoms A and Bouckoms A (1985). Affective disturbance and denial of problems in dental patients with pain. Int. J. Psychosom. 32:9-11.

Arnstein P, Caudill M, Mandle CL, Norris A and Beasley R (1999). Self efficacy as a mediator of the relationship between pain intensity, disability and depression in chronic pain patients. Pain 80:483-491.

Arntz A, van Eck M and Heijmans M (1990). Predictions of dental pain: the fear of any expected evil is worse than the evil itself. Behav. Res. Ther. 28:29-41.

Asmundson GJG, Norton PJ and Veloso F (1999). Anxiety sensitivity and fear of pain in patients with recurring headaches. Behav. Res. Ther. 37:703-713.

Auerbach SM, Laskin DM, Frantsve LM and Orr T (2001). Depression, pain, exposure to stressful life events, and long-term outcomes in temporomandibular disorder patients. J Oral Maxillofac. Surg. 59:628-633.

Babor T and Grant M (1989). From clinical research to secondary prevention: International collaboration in the development of the Alcohol Use Disorders Identification Test (AUDIT). Alc. Health Res. World 13:371-374.

Bade DM & Dimitroff M (1990). Personality characteristics in the TMJ pain patient. CDS Rev 83:26-31.

Bagby RM, Taylor GJ & Parker JD (1994). The Twenty-item Toronto Alexithymia Scale--II. Convergent, discriminant, and concurrent validity. Psychosom Res 38:33-40.

Baggi L, Rubino IA, Zanna V and Martignoni M (1995). Personality disorders and regulative styles of patients with temporo-mandibular joint pain dysfunction syndrome. Percept. Motor Skills 80:267-273.

Bahra A and Goadsby P (1998). Co-morbidity of migraine and depression: is there a link? Progress Neurol. Psychiat. 2:23-27.

Bakal D (1997). Clinical complexities of managing pain, suffering, and analgesic dependence. Headache Quarterly 8:137-149.

Bandura A (1977). Social Learning Theory. Prentice, Englewood Cliffs, NJ.

Bandura A (1974). Behaviour theory and models of man. Amer. Psychol. 29:859-869.

Banks S and Kerns R (1996). Explaining high rates of depression in chronic pain: a diathesis-stress framework. Psychol. Bull. 119:95-110.

Barkwell DP (1991). Ascribed meaning: a critical factor in coping and pain attenuation in patients with cancer-related pain. J. Pall. Care 7:5-14.

Baron R, Logan H and Hoppe S (1993). Emotional and sensory focus as mediators of dental pain among patients differing in desired and felt dental control. Health Psychol. 12:381-389.

Bates M, Edwards W & Anderson K (1993). Ethnocultural influences on variation in chronic pain perception. Pain 52:101-112.

Baume R, Croog S and Nalbandian J (1995). Pain perception, coping strategies, and stress management among periodontal patients with repeated surgeries. Percept. Motor Skills 80:307-319.

Beck A (1976). Cognitive therapy and the emotional disorders. Int. Uni. Press, NY

Beck A, Ward C, Mendelson M, Mock J and Erbaugh J (1961). An inventory for measuring depression. Arch. Gen. Psychiat. 4:561-571.

Beck A and Weissman A (1974). The measurement of pessimism. The hopelessness scale. J. Consult. Clin. Psychol. 42:361-365.

Beckham JL, Keefe FJ, Caldwell DS & Roodman AA (1991). Pain coping strategies in rheumatoid arthritis: relatonships to pain, disability, depression and daily hassles. Behav. Ther. 22:113-124.

Bergner M and Bobbitt R (1981). The Sickness Impact Profile: Development and final revision of a health status measure. Med. Care 19:787-805.

Bertram S, Rudisch A, Innerhofer K, Pumpel E, Grubweiser G and Emshoff R (2001). Diagnosing TMJ internal derangement and osteoarthritis with magnetic resonance imaging. J Amer. Dent. Assoc. 132:753-761.

Bianchi P, Cacchiotti D, McNeill C et al (1992). MMPI results comparing craniofacial pain/TMJ patients and non patients. J Dent Res: AADR Abstract 1083.

Blalock SJ, DeVellis BM & DeVellis RF (1989). Social comparison among individuals with rheumatoid arthritis. J. Appl. Soc. Psychol. 19:665-680.

Blomkvist V, Hannerz J, Orth-Gomer K and Theorell T (1997). Coping style and social support in women suffering from cluster headache or migraine. Psychother. Psychosom. 66:150-154.

Blumer D & Heilbronn M (1982). Chronic pain as a variant of depressive disease: the pain-prone disorder. J Nervous & Mental Dis. 170:381-394.

Bonica JJ (1980). Pain Research Therapy. Past and current status and future needs. pp.1-48. Elsever, Amsterdam.

Borkovec T and O'Brien G (1977). Relation of autonomic pereception and its manipulation to the maintenance and reduction of fear. J. Abnorm. Psychol. 86:163-171.

Bosmajian P (1981). Comparison of stress inoculation training and stress education in the treatment of stress associated with dental procedures. Dissert. Abstr. Int. 42:4181B.

Bradley PF (1987). Conservative treatment for temporomandibular joint pain

dysfunction. Br J Oral Maxillofac Surg 25:125-137.

Breslau N (1992). Migraine, suicide ideation and suicide attempts. Neurology 42:392-395.

Breslau N, Davis G, Schultz L and Patterson E (1994). Migraine and major depression. Headache 34:387-393.

Breslau N, Chilcoat H and Andreski P (1996). Further evidence of the link between migraine and neuroticism. Neurology 47:663-667.

Brooke RI and Stenn PG (1983). Myofascial pain dysfunction syndrome - how effective is biofeedback-assisted relaxation training? In: JJ Bonica et al (Eds.). Advances in Pain Research and Therapy. Raven Press, New York, pp.809-812.

Brook R, Ware J, Davies-Avery A et al (1979). Conceptualization and Measurement of Health for Adults in the Health Insurance Study. Vol.8: overview. Rand Corporation, Santa Monica, CA.

Brown G (1990). A causal analysis of chronic pain and depression. J. Abnorm. Psychol. 99:127-137.

Brown GK, & Nicassio PM (1987). The development of a questionnaire for the assessment of active and passive coping strategies in chronic pain patients. Pain 31:53-65.

Brown GK, Nicassio PM & Wallston KA (1989). Pain coping strategies and depression in rheumatoid arthritis. J. Consult. Clin. Psychol. 57:652-657.

Browne G, Arpin K, Corey P et al (1990). Individual correlates of health service utilization and the cost of poor adjustment to chronic illness. Med. Care 28:43-58.

Buckelew SP, Conway RC, Shutty MS, Lawrence JA, Grafting MR, Anderson SK, Hewett JE & Keefe FJ (1992). Spontaneous coping strategies to manage acute pain and anxiety during electrodiagnostic studies. Arch. Phys. Med. Rehab. 73:594-598.

Buckelew S, Parker J, Keefe F, Deuser W, Crews T, Conway R, Kay D & Hewett J (1994). Self-efficacy and pain behavior among subjects with fibromyalgia. Pain 59:377-384.

Buckelew S, Shutty M, Hewett J, Landon T, Morrow K & Frank R (1990). Health locus of control, gender differences and adjustment to persistent pain. Pain 42:287-294.

Buckingham RB, Braun T, Harinstein DA et al (1991). Temporomandibular joint dysfunction syndrome: a clase association with systemic joint laxity (the hypermobile joint syndrome) Oral Surg, Oral Med, Oral Path 72:514-519.

Burckhardt C & Bjelle A (1994). A Swedish version of the short-form McGill Pain Questionnaire. Scand J Rheumatol 23:77-81.

Burckhardt C, Mannerkorpi K, Hedenberg L and Bjelle A (1994). A randomized, controlled clinical trial of education and physical training for women with fibromyalgia. J. Rheumatol. 21:714-720.

Burns JW, Johnson BJ, Devine J, Mahoney N and Pawl R (1998). Anger management style and the prediction of treatment outcome among male and female chronic pain patients. Behav. Res. Ther. 36:1051-1062.

Bush F and Harkins S (1995). Pain-related limitations in activities of daily living in patients with chronic orofacial pain: Psychometric properties of a disability index. J. Orofac. Pain 9:57-63.

Byrne D (1961). The repression-sensitization scale: Its reliability and validity. J. Pers. 9:334-338.

Carosella A, Lackner J & Feuerstein M (1994). Factors associated with early discharge from a multidisciplinary work rehabilitation program for chronic low back pain. Pain 57:69-76.

Carr AB, Christensen LV, Donegan SJ & Ziebert GJ (1991). Postural contractile activities of human jaws muscles following use of an occlusal splint. J Oral Rehab 18:185-191.

Casten R, Parmelee P, Kleban M, Lawton P and Katz I (1995). The relationships among anxiety, depression, and pain in a geriatric institutionalized sample. Pain 61:271-276.

Cattell R and Eber H (1964). Handbook for the 16 Personality Factor Questionnaire. Institute for Personality and Ability Testing, Champaign, IL.

Centore L & McNeill C (1992). The relationship between non-organic multiple physical complaints and narcissism. J Dent Res: AADR Abstract 1084.

Cerbo R, Barbanti P, Fabbrini G, Pascali MP and Cartarci T (1998). Amitriptyline is effactive in chronic but not in episodic tension-type headache: Pathogenetic implications. Headache 38:453-457.

Chapman S (1986). A review and clinical perspective on the use of EMG and thermal biofeedback for chronic headaches. Pain 27:1-43.

Chaves J and Brown J (1987). Spontaneous cognitive strategies for the control of clinical pain and stress. J. Behav. Med. 10:263-276.

Cimino R, Michelotti A, Stradi R and Farinaro C (1998). Comparison of clinical and psychologic features of fibromyalgia and masticatory myofascial pain. J. Orofac. Pain;12:35-41.

Clark GT, Green EM, Dorman MR & Flack VF (1987). Craniocervical dysfunction levels in a patient sample from a temporomandibular joint clinic. J Amer Dent Assoc 115:251-256.

Clark GT, Adachi NY & Dornan MR (1990). Physical medicine procedures affect temporomandibular disorders: a review. J Amer Dent Assoc 121:151-161.

Cohen S, Kamarck T & Mermelstein R (1983). A global measure of perceived stress. J. Health Social Behav. 24:385-396.

Coia P and Morley S (1998). Medical reassurance and patients' responses. J. Psychosom. Res. 45:377-386.

Connor E and Jeffrey I (1984). An investigation into dental pain. Dent. Update 11:644-652.

Corah N (1969). Development of a dental anxiety scale. J. Dent. Res. 48:596.

Corah N, O'Shea R, Bissell D et al (1978). The dentist-patient relationship: perceived dentist behaviors that reduce patient anxiety and increase satisfaction. J. Amer. Dent. Ass. 116:73-76.

Costa PT and McCrae RR (1985). The NEO Personality Inventory Manual. Odessa, FL: Psychological Assessment Resources.

Cott A, Parkinson W, Fabich M, Bedard M and Marlin R (1992). Long-term efficacy of combined relaxation: Biofeedback treatments for chronic headache. Pain 51:49-56.

Council J, Ahem D, Follick M & Kline C (1988). Expectancies and functional impairment in chronic low back pain. Pain 33:323-331.

Crisson J & Keefe F (1988). The relationship of locus of control to pain coping strategies and psychological distress in chronic pain patients. Pain 35:147-154.

Croog S, Baume R and Nalbandian J (1994). Pain response after psychological preparation for repeated periodontal surgery. J. Amer. Dent Ass. 125:1353-1360.

Croog S, Baume R and Nalbandian J (1995). Pre-surgery psychological characteristics, pain response, and activities impairment in female patients with repeated periodontal surgery. J. Psychosom. Res. 39:39-51.

Crowne D and Marlowe D (1960). A new scale of social desirability independent of psychopathology. J. Consult. Psychol. 24:349-354.

Curtis E (1990). The string and the doorknob: profile of a popular approach to dental extraction. J. Oral Maxillofac. Surg. 48:1084-1092.

Dahlstrom L, Kahnberg K-E & Lindahl L (1989). 15 years follow-up on condylar fractures. Int J Oral Maxillofac Surg 18:18-23.

Dao T, Lund J and Lavigne G (1994). Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. J. Orofac. Pain 8:350-355.

Davis P, Holm J, Myers T and Suda K (1998). Stress, headache, and phsiological disregulation: A time-series analysis of stress in the laboratory. Headache 38:116-121.

De Benedettis G and Lorenzetti A (1992). Minor stressful life events (daily hassles) in chronic primary headache: Relationship with MMPI personality patterns. Headache 32:330-332.

de Bruijn-Kofman A, van der Wiel H, Groenman N, Sorbi M and Klip E (1997). Effects of a mass media behavioral treatment for chronic headcahes. Haedache 37:415-420.

de Jongh A, Muris P, ter Horst G et al (1995). Acquisiton and maintenance of dental anxiety: the role of conditioning experiences and cognitive factors. Behav. Res. Ther. 33:205-210.

De Kanter EJ, Kayser AF, Battistuzzi PG et al (1992). Demand and need for treatment of craniomandibular dysfunction in the Dutch adult population. J Dent Res 71:1607-1612.

Demjen S, Bakal D and Dunn B (1990). Cognitive correlates of headache intensity and duration. Headache 30:423-427.

DeVellis RF, Holt K, Renner BR, Blalock SJ, Blanchard LW, Cook HL, Koltz ML Mikov V & Harring K (1990). The relationship of social comparison to rheumatoid arthritis symptoms and affect. Basic Appl. Soc. Psychol. 11:1-18.

Di Fabio R (1998). Physical therapy for patients with TMD: A descriptive study of treatment, disability, and health status. J. Orofac. Pain 12:124-135.

Dolce J, Crocker M, Moletteire C et al (1986). Exercise quotas, anticipatory concern and self-efficacy expectancies in chronic pain; a preliminary report. Pain 24:365-372.

Dohrenwend BP, Raphael KG, Marbach JJ and Gallagher RM (1999). Why is depression comorbid with chronic myofascial face pain? A family study test of alternative hypotheses. Pain 82:1-11.

Dozois D, Dobson K, Wong M, Hughes D and Long A (1996). Predictive utility of the CSQ in low back pain: individual vs. composite measures. Pain 66:171-180.

Dworkin R, Hartstein G, Rosner H et al (1992). A high-risk method for studying psycho-social antecedents of chronic pain: the prospective investigation of herpes zoster. J Abn. Psychol. 101:200-205.

Dworkin S and LeResche L (1992). Research diagnostic criteria for temporomandibular disorders: review, criteria, examination and specifications, critique. J. Craniomandib. Disord. 6:301-355.

Dworkin SF, Turner JA, Wilson L, Massoth D, Whtiney C, Huggins KH et al (1994). Brief group cognitive-behavioral intervention for temporomandibular disorders. Pain 59:175-187.

Dworkin S, LeResche L, DeRouenT and von Korff M (1990). Assessing clinical signs of temporomandibular disorders: Reliability of clinical examiners. J. Prosthet. Dent.63:574-579.

Dworkin S and Massoth D (1994). Temporomandibular disorders and chronic pain: Disease or illness? J Prosthet. Dent. 72:29-38.

Eccleston C and Crombez G (1999). Pain demands attention: A cognitive-affective model of the interruptive function of pain. Psychol. Bull. 125:356-366.

Edmeads J (1998). Why is migraine so common? Cephalalgia 18 Supp.22:2-4.

Edwards RR, Fillingim RB, Yamauchi S, Sigurdsson A, Bunting S, Mohorn SG & Maixner W (1999) Effects of gender and acute dental pain on thermal pain responses. Clin J Pain 15:233-7.

Edwards L, Pearce S, Turner-Stokes L and Jones A (1992). The Pain Beliefs Questionnaire: an investigation of belief in the causes and consequences of pain. Pain 51:267-272.

Eli I, Uziel N, Baht R et al (1997). Antecedents of dental anxiety: learned responses versus personality traits. Comm. Dent. Oral Epidemiol. 25:233-237.

Ellertsen B, Nordby H and Sjaastad O (1987). Psychophysiologic response patterns in tension headache: Effects of tricyclic antidepressants. Cephalalgia 7:55-63.

Ellis A and Bernard M (1986). What is rational emotive therapy (RET)? In: Handbook of rational emotive therapy, vol. 2. Ellis A, Grieger RM (eds). Springer Publishing Co., New York.

Endler N and Parker D (1990). Coping Inventory for Stressful Situations (CISS): Manual. Multi-Health Systems, Toronto.

Endler NS, Parker JDA and Summerfeldt LJ (1992). Coping with Health Problems: Developing a reliable and valid multidimensional measure. Dept. Psychol. Reports No. 204, York University, Toronto.

Enqvist B and Fischer K (1997). Preoperative hypnotic techniques reduce consumption of analgesics after surgical removal of third mandibular molars: a brief communication. Int. J. Clin. Exp. Hypn. 45:102-108.

Epker J and Gatchel RJ (2000). Prediction of treatment-seeking behavior in acute TMD patients: practical application in clinical settings. J Orofac Pain 14:303-309.

Epker J, Gatchel RJ and Ellis E III (1999). A model for predicting chronic TMD: Practical application in clinical settings. J. Amer. Dent. Ass. 130:1470-1475.

Epstein JB, Caldwell J and Black G (2001). The utility of panoramic imaging of the temporomandibular joint in patients with temporomandibular disorders. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 92:236-239.

Estlander A-M, Takala EP and Verkasalo M (1995). Assessment of depression in chronic musculoskeletal pain patients. Clin. J Pain 11:194-200.

Eysenck H and Eysenck B (1964). Manual of the Eysenck Personality Inventory. London.

Eysenck H and Eysenck S (1968). Manual of the Eysenck Personality Inventory. Educational and Industrial Testing Service, San Diego, CA.

Eysenck H and Eysenck S (1975). Manual of the Eysenck Personality Questionnaire.University of London, Hodder and Stoughton.

Faucett J, Gordon N and Levine J (1994). Differences in postoperative pain severity among four ethnic groups. J. Pain Sympt. Manage. 9:383-389.

Fearon C & Serwalka W (1983). Stress: a common denominator for non-organic TMJ pain dysfunction. J Pros Dent 49:805.

Feinmann C (1985). Pain relief by antidepressants: possible modes of action. Pain 23: 1-8.

Feinmann C & Harris M (1984). Psychogenic facial pain. Part 2. Management and prognosis. Br. Dent. J 156:205-212.

Feinmann C, Ong M, Harvey W and Harris M (1987). Psychological factors influencing post-operative pain and analgesic consumption.. Br. J. Oral Maxillofac. Surg. 25:285-292.

Ferguson RJ and Ahles TA (1998). Private body consciousness, anxiety and pain symptom reports of chronic pain patients. Behav. Res. Ther. 36:527-535.

Fernandez E and Sheffield J (1996). Relative contributions of life events versus daily hassles to the frequency and intensity of headaches. Headache 36:595-602.

Ferrari R & Leonard MS (1998). Whiplash and temporomandibular disorders: a critical review. J Amer Dent Assoc 129:1739-1745.

Ferrer-Perez VA, Truyols Tabemer MM (1996). The role of coping strategies in patients with benign chronic pain. Abstracts 8th World Congress on Pain 72.

Fifield J, Reisine S & Grady K (1991). Work disability and the experience of pain and depression in rheumatoid arthritis. Soc. Sci. Med. 33:579-585.

Fiset L, Leroux B, Rothen M, Prall C, Zhu C and Ramsay D (1997). Pain control in recovering alcoholics: effects of local anesthesia. J. Stud. Alcohol 58:291-296.

Fishbain D, Goldberg M, Meagher B et al (1986). Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. Pain 26:181-197.

Forssell H, Kalso E, Koskela P, Vehmanen R, Puukka P and Alanen P (1999). Occlusal treatments in temporomandibular disorders: a qualitative systematic review of randomized controlled trials. Pain 83:549-560.

Forward S, McGrath P, MacKinnon D, Brown T, Swann J and Currie E (1998). Medication patterns of recurrent headache sufferers: A community study. Cephalalgia 18:146-151.

Fries J, Carey C and McShane D (1997). Patient education in arthritis: Randomized controlled trial of a mail-delivered program. J. Rheumatol. 24:1378-1383.

Funch D & Gale E (1985). Predicting treatment completion in a behavioral therapy program for chronic temporomandibular pain. J Psychosom. Res. 30:57-62.

Gardea MA, Gatchel RJ and Mishra KD (2001). Long-term efficacy of biobehavioral treatment of temporomandibular disorders. J Behav. Med. 24:341-359.

Gatchel RJ, Garofalo J, Ellis E and Holt C (1996). Major psychological disorders in acute and chronic TMD: an initial examination. J. Amer. Dent. Ass. 127: 1365-1374.

Gentry M (1997). Third molar extraction; the effects of a cognitive pain coping strategy on pre-surgical anxiety and post-surgical pain and coping. Unpublished MSc. thesis, University College London.

Geer J (1965). In: Geer J (1966). Fear and autonomic arousal. J Abnorm Psychol 71:253-255.

George J, Scott D, Turner S and Gregg J (1980). The effects of psychological factors and physical trauma on recovery from oral surgery. J. Behav. Med. 3:291-310.

Gessel AH (1975). Electromyographic biofeedback and tricyclic antidepressants in myofascial pain dysfunction syndrome: psychological predictors of outcome. J Amer Dent Assoc 91:1048-1052.

Getka E and Glass C (1992). Behavioral and cognitive-behavioral approaches to the reduction of dental anxiety. Behav. Ther. 23:433-448.

Gidron Y, McGrath P and Goodday R (1995). The physical and psychosocial predictors of adolescents' recovery from oral surgery. J. Behav. Med. 18:385-399.

Gil K, Abrams M, Phillips G and Keefe F (1989). Sickle cell disease pain: Relation of coping strategies to adjustment. J. Consult. Clin. Psychol. 57:725-731.

Gil KM, Williams DA, Thompson RJ Jr & Kinney TR (1991). Sickle cell disease in children and adolescents: the relation of child and pain coping strategies to adjustment. J. Paed. Psychol. 16:643-663.

Glaros AG, Glass EG & McLaughlin L (1994). Knowledge and beliefs of dentists regarding temporomandibular disorders and chronic pain. J Orofac Pain 8:216-222.

Glaros AG, Forbes M, Shanker J and Glass EG (2000). Effect of parafunctional clenching on TMD pain and proprioceptive awareness. Cranio 18:198-204.

Gobel H, Hamouz V, Hansen C, Heininger K, Hirsch S, Lindner V, Heuss D and Soyka D (1994). Chronic tension-type headache: Amitriptyline reduces clinical headache-duration and experimental pain sensitivity but does not alter pericranial muscle activity readings. Pain 59:241-249.

Goldberg D (1972). The Detection of Psychiatric Illness by Questionnaire. OUP, London.

Goldberg R (1994). Childhood abuse, depression and chronic pain. Clin. J. Pain 10:277-281.

Gotlib I, Lewinsohn P and Seeley J (1995). Symptoms versus diagnosis of depression: Differences in psychosocial functioning. J. Consult. Clin. Psychol. 63:90-100.

Gracely R and Kwilosz D (1988). The descriptor differential scale: Applying psychophysical principles to clinical pain assessment. Pain 35:279-288.

Gratt BM & Sickles EA (1993). Thermographic characterisation of the asymptomatic temporomandibular joint. J Orofac Pain 7:7-14.

Graff-Radford S and Solberg W (1992). Atypical odontalgia. J. Craniomandib. Disord. 6:260-5.

Graham JR (1990). The Minnesota Multiphasic Personality Inventory 2: Assessing personality and psychopathology. New York: Oxford University Press.

Granella F, Farina S, Malferrari G and Manzoni GC (1987). Drug abuse in chronic headache: A clinico-epidemiologic study. Cephalalgia 7:15-19.

Greene CS (1992). Managing TMD patients: initial therapy is the key. J Amer Dent Assoc 123:43-45.

Greene CS & Laskin DM (1972). Splint therapy for the myofascial pain dysfunction (MPD) syndrome: a comparative study. J Amer Dent Assoc 84:624-628.

Greene CS & Laskin DM (1988). Long term status of TMJ clicking in patients with myofascial pain and dysfunction. J Amer Dent Asoc 172:461-465.

Gross AR (1986). The effect of coping strategies on the relief of pain following surgical interventions for lower back pain. Behav. Ther. 21:349-352.

Gross P (1992). Is pain associated with dental avoidance? Behav. Res. Ther. 30:7-13.

Guidetti V, Gaali F, Fabrizi P, Giannantoni AS, Napoli L, Bruni O and Trillo S (1998). Headache and psychiatric comorbidity: Clinical aspects and outcome of an 8-year follow-up study. Cephalalgia 18:455-462.

Hadjistavropoulos HD and Asmundson GJG (1998). Factor analytic investigation of the Illness Attitudes Scale in a chronic pain sample. Behav. Res. Ther. 36:1185-1195.

Hadjistavropoulos HD, Asmundson GJG and Norton GR (1998). Validation of the Coping with Health, Injuries and Problems scale in a chronic pain sample. Clin. J. Pain 15:41-49.

Hadjistavropoulos HD, McLeod FK and Asmundson GJG (1999). Validation of the Chronic Pain Coping Inventory. Pain 80:471-481.

Hagglund KJ, Haley WE, Reveille JD & Alarcon GS (1989). Predicting individual differences in pain and functional impairment among patients with rheumatoid arthritis. Arth. Rheum. 32:851-858.

Hameroff S, Cork R, Scherer K et al (1982). Doxepin effects on chronic pain, depression and plasma opioids. J Clin. Psychiat. 43:22-27.

Hansson P, Ekblom A, Thomsson M and Fjellner B (1989). Pain development and consumption of analgesics after oral surgery in relation to personality characteristics. Pain 37:271-277.

Hargreaves K, Dionne R and Mueller G (1983). Plasma beta-endorphin-like immunoreactivity, pain and anxiety following administration of placebo in oral surgery patients. J. Dent. Res. 62:1170-1173.

Harkins S, Linford J, Cohen J, Kramer T & Cueva L (1991). Administration of clonazepam in the treatment of TMD and associated myofascial pain: a double-blind pilot study. J Craniomandib Disord Fac Oral Pain 5:179-186.

Harkins SJ & Marteney JL (1980). Extrinsic trauma: a significant precipitating factor in temporomandibular pain syndrome. J Pros Dent 44:642-653.

Harrison S, Glover L, Maslin L, Feinmann C, Pearce S and Harris M (1997). A comparison of antidepressant medication alone and in conjunction with cognitive behavioral therapy for chronic idiopathic facial pain. Proceedings of the 8th World Congress on Pain: Progress in Pain Research and Management, Vol.8, eds. Jensen T, Turner J & Weisenfeld-Hallin Z, IASP Press, Seattle,WA.

Hatch J, Schoenfeld L, Boutros N, Seleshi E, Moore P and Cyr-Provost M (1991). Anger and hostility in tension-type headache. Headache 31:302-304.

Haythomthwaite JA, Menefee LA, Heinberg LJ and Clark MR (1998). Pain coping strategies predict perceived control over pain. Pain 77:33-39.

Haythomthwaite J, Sieber W & Kerns R (1991). Depression and the chronic pain experience. Pain 46:177-184.

Heijmans M (1998). Coping and adaptive outcome in chronic fatigue syndrome: Importance of illness cognitions. J. Psychosom. Res. 45:39-51.

Heitkemper T, Layne C and Sullivan D (1993). Brief treatment of children's dental pain and anxiety. Percept. Motor Skills 76:192-194.

Hendler N (1984). Depression caused by chronic pain. J Clin. Psychiat. 45:30-36.

Herda C, Siegeris K & Basler H-D (1994). The Pain Beliefs and Perceptions Inventory: further evidence for a 4-factor structure. Pain 57:85-90.

Hill A, Niven C and Knussen C (1995). The role of coping in adjustment to phantom limb pain. Pain 62:79-86.

Ho K-H, Ong B and Lee S-C (1997). Headache and self-assessed depression scores in Singapore University undergraduates. Headaches 37:26-30.

Holm J, Bury L and Suda K (1996). The relationship between stress, headache, and the menstrual cycle in young female migraineurs. Headache 36:531-537.

Holm J, Lamberty K, McSherry W II and Davis P (1997). The stress response in headache sufferers: Physiogical and psychological reactivity. Headache 37:221-227.

Holmes JA & Stevenson CAZ (1990). Differential effects of avoidant and attentional coping strategies on adaption to chronic and recent-onset pain. Health Psychol. 9:577-584.

Holroyd KA, Nash J, Pingel J, Cordingley G and Jerome A (1991). A comparison of pharmacological (amitriptyline HCl) and nonpharmacological (cognitive-behavioral) therapies for chronic tension headaches. J. Consult. Clin. Psychol. 59:387-393.

Holroyd KA, O'Donnell FJ, Stensland M, Lipchik GL, Cordingley GE and Carlson BW (2001). Management of chronic tension-type headache with tricyclic antidepressant medication, stress management therapy, and their combination: a randomized controlled trial. J Amer. Med. Assoc. 285:2208-2215.

Holroyd KA and Penzien D (1990). Pharmacological versus non-pharmacological prophylaxis of recurrent migraine headache: A meta-analytic review of clinical trials. Pain 42:1-13.

Holzberg A, Robinson M & Geisser M (1993). The relationship of cognitive distortion to depression in chronic pain: the role of ambiguity and desirability in self-ratings. Clin. J Pain 9:202-206.

Horan J, Layng F and Pursell C (1976). Preliminary study of effects of 'in vivo' emotive imagery on dental discomfort. Percept. Motor Skills 12:105-106.

Houle M, McGrath P, Moran G and Garrett O (1988). The efficacy of hypnosis- and relaxation-induced analgesia on two dimensions of pain for cold pressor and electrical tooth pulp stimulation. Pain 33:241-251.

Humphris G, Mair L, Lee G and Birch R (1991). Dental anxiety, pain and uncooperative behaviour in child dental patients. Anxiety Res. 4:61-77.

Hursey K and Jacks SD (1992). Fear of pain in recurrent headache sufferers. Headache 32:283-286.

Husek T and Alexander S (1963). The effectiveness of the Anxiety Differential in examination stress situations. Educ. Psychol. Measure 23:309-318.

International Headache Classification Committee (1997). ICD-10 Guide for Headaches. Cephalalgia 17 Suppl.19:1-82.

Ishigaki S, Bessette RW & Maruyama T (1993). Vibration analysis of the temporomandibular joints with meniscal displacement with and without reduction. J Craniomandib Pract 11:192-201.

Jagger RG (1973). Diazepam in the treatment of temporomandibular joint dysfunction syndrome - a double blind study. J Dent 2:37-40.

Janssen S & Arntz A (1996). Anxiety and pain: attentional and endorphinergic influences. Pain 66:145-150.

Jaspers JP, Heuvel F, Stegenga B & de Bont LG (1993). Strategies for coping with pain and psychological distress associated with temporomandibular joint osteoarthrosis and internal derangement. Clin. J. Pain 9:94-103.

Jelencsik I (1998). Primary headache and its contemporary management. Orv. Hetil. 139:1723-1728.

Jensen I, Nygren A, Gamberale F, Goldie I & Westerholm P (1994). Coping with long-term musculoskeletal pain and its consequences: is gender a factor? Pain 57:167-172.

Jensen MP & Karoly P (1991). Control beliefs, coping efforts and adjustment to chronic pain. J. Consult. Clin. Psychol. 59:431-438.

Jensen MP & Karoly P (1991). Comparative self-evaluation and depressive affect among chronic pain patients. Quoted in: Jensen et al(1991).

Jensen MP, Karoly P & Huger R (1987). The development and preliminary validation of an instrument to assess patients' attitudes towards pain. J Psychosom. Res. 31:393-400.

Jensen MP, Nielson WR, Romano JM, Hill ML & Turner JA (2000). Further evaluation of the pain stages of change questionnaire: is the transtheoretical model of change useful for patients with chronic pain? Pain 86:255-264.

Jensen MP, Romano JM, Turner JA, Good AB and Wald LH (1999). Patient beliefs predict patient functioning: Further support for a cognitive-behavioural model of chronic pain. Pain 81:95-104.

Jensen MP, Turner JA & Romano JM (1994). Correlates of improvement in multidisciplinary treatment of chronic pain. J Consult. Clin. Psychol. 62:172-179.

Jensen MP, Turner JA and Romano JM (2001). Changes in beliefs, catastrohizing, and coping are associated with improvement in multidisciplinary pain treatment. J Consult. Clin. Psychol. 69:655-662.

Jensen MP, Turner JA, Romano JM & Karoly P (1991). Coping with chronic pain: a critical review of the literature. Pain 47:249-283.

Jensen MP, Turner JA, Romano JM and Lawler BK (1994). Relationship of pain-specific beliefs to chronic pain adjustment. Pain 57:301-309.

Jensen MP, Turner JA, Romano JM & Strom S (1995). The Chronic Pain Coping Inventory: development and preliminary validation. Pain 60:203-216.

Jerusalem M and Schwarzer R (1992) Self-efficacy as a resource factor in stress appraisal processes. In: Schwarzer R et al (Eds). Self-efficacy: Thought control of action. (pp. 195-213). Hemisphere Publishing Corp.: Washington, DC, USA.

Johansson F & von Knorring L (1979).A double-blind controlled study of a serotonin uptake inhibitor (Zimelidine) versus placebo in chronic pain patients. Pain 7:69-78.

Johansson P and Berggren U (1992). Assessment of dental fear: a comparison of two psychometric instruments. Acta Odontol. Scand. 50:43-49.

Jones M and Chan C (1992). The pain and discomfort experienced during orthodontic treatment: a randomized controlled clinical trial of two initial aligning arch wires. Amer. J. Orthod. Dentofac. Orthop. 102:373-381.

Kampe T & Hannerz H (1991). Five year longitudinal study of adolescents with intact and restored dentitions: signs and symptoms of temporomandibular dysfunction and functional recordings. J Oral Rehab 18:387-398.

Katcher A, Segal H and Beck A (1984). Comparison of contemplation and hypnosis for the reduction of anxiety and discomfort during dental surgery. Amer. J. Clin. Hypn. 27:14-21.

Keefe FJ, Caldwell DS, Queen KT, Gil KM, Martinez S, Crisson JE, Ogden W & Nunley J (1987a). Osteoarthritic knee pain: a behavioral analysis. Pain 28:309-321.

Keefe FJ, Caldwell DS, Queen KT, Gil KM, Martinez S, Crisson JE, Ogden W & Nunley J (1987b).Pain coping strategies in osteoarthritis patients. J. Consult. Clin. Psychol. 55:208-212.

Keefe FJ, Caldwell DS, Williams DA, Gil KM, Mitchell D, Marrtinez S, Nunley J, Beckham JC, Crisson JE & Helms M (1990a). Pain coping skills training in the management of osteoarthritic knee pain: a comparative study. Behav. Ther. 21:435-437.

Keefe FJ, Crisson JE, Urban BJ & Williams DA (1990). Analyzing chronic low back pain: the relative contribution of pain coping strategies. Pain 40:293-301.

Keefe FJ, Lefebvre JC, Kerns RD, Rosenberg R, Beaupre P, Prochaska J, Prochaska JO & Caldwell DS (2000). Understanding the adoption of arthritis self-management: stages of change profiles among arthritis patients. Pain 87:303-313.

Keefe FJ, Salley AN & Lefebvre JC (1992). Coping with pain: conceptual concerns and future directions. Pain 51:131-134.

Keefe FJ & Williams DA (1990). A comparison of coping strategies in chronic pain patients in different age groups. J. Gerontol. 45:161-165.

Kellner R (1986). Somatization and Hypochondriasis. New York: Praeger.

Kent G (1984). Anxiety, pain and type of dental procedure. Behav. Res. Ther. 22:465-469.

Kent G (1985). Memory of dental pain. Pain 21:187-194.

Kent G (1986). The typicality of therapeutic 'surprises'. Behav. Res. Ther. 24:625-628.

Kent G (1987). Self-efficaceous control over reported physiological, cognitive and behavioural symptoms of dental anxiety. Behav. Res. Ther. 25:341-347.

Kerns R, Rosenberg R, Jamison R, Caudill M and Haythornthwaite J (1997). Readiness to adopt a self-management approach to chronic pain: the Pain Stages of Change Questionnaire (PSOCQ). Pain 72:227-24.

Kersh BC, Bradley LA, Alarcon GS, Alberts KR, Sotolongo A, Martin MY, Aaron LA, Dewaal DF, Domino ML, Chaplin WF, Palardy NY, Cianfrini LR, Triana-Alexander M (2001). Psychosocial and health status variables independently predict health care seeking in fibromyalgia. Arthritis Reum. 45:362-371.

Kight M, Gatchel RJ and Wesley LaV (1999). Temporomandibular disorders: Evidence for significant overlap with psychopathology. Health Psychol. 18:177-182.

Kinney R, Gatchel R, Ellis E and Holt C (1992). Major psychological disorders in chronic TMD patients: Implications for successful management. J. Amer. Dent. Assoc. 123:49-54.

Kirk WS & Calabrese DK (1989). Clinical evaluation of physical therapy in the management of internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 47:113-119.

Kleinknecht R and Bernstein D (1978). The assessment of dental fear. Behav. Ther. 9:626-634.

Kleinknecht R, Klepac R and Alexander L (1973). Origins and characteristics of fear of dentistry. J. Amer. Dent. Ass. 86:842-848.

Klepac R, Dowling J and Hauge G (1982). Characteristics of clients seeking therapy for the reduction of dental avoidance: reactions to pain. J. Behav. Ther. Exp. Psychiatr. 13:293-300.

Klepac R, McDonald M, Hauge G and Dowling J (1980). Reactions to pain among subjects high and low in dental fear. J. Behav. Med. 3:373-383.

Kohler T and Kosanic S (1992). Are persons with migraine characterized by a high degree of ambition, orderliness and rigidity? Pain 48:321-323.

Kores R, Murphy W, Rosenthal T, Elias D & North W (1990). Predicting outcome of chronic pain treatment via a modified self-efficacy scale. Beh.Res.Ther. 28:165-169.

Korszun A, Hinderstein B and Wong M (1996). Comorbidity of depression with chronic facial pain and temporomandibular disorders. Oral Surg. Oral Med. Oral Pathol. 82:496-500.

Koutantji M, Pearce S and Oakley D (1998). The relationship between gender and family history of pain with current pain experience and awareness of pain in others. Pain 77:25-31.

Kreitler H & Kreitler S (1976) eds: Cognitive Orientation and Behavior. Springer, NY.

Kreitler S, Kreitler H & Carasso R (1987). Cognitive orientation as predictor of pain relief following acupuncture. Pain 28:323-341.

Krishnan K, France R, Pelton S, McCann U, Davidson J and Urban B (1985). Chronic pain and depression II. Symptoms of anxiety in chronic low back pain patients and their relationship to subtypes of depression. Pain 22:289-294.

Kropp P, Gerber-Wolf D, Keinath-Specht A, Kopal T and Niederberger U (1997). Behavioral treatment in migraine. Cognitive-behavioral therapy and blood-volume-pulse biofeedback: A cross-over study with a two-year followup. Funct. Neurol: New Trends Adapt. Behav. Disord. 12:17-24.

Kunzelmann K-H and Dunninger P (1990). Dental fear and pain: effect on patient's perception of the dentist. Comm. Dent. Oral Epidemiol. 18:264-266.

Kurita H, Ohtsuka A, Kurashina K and Kopp S (2001). Chewing abiliaty as a parameter for evaluating the disability of patients with temporomandibular disorders. J Oral Rehab. 28:463-465.

Kurtze N, Gundersen KT and Svebak S (1998). The role of anxiety and depression in fatigue and patterns of pain among subgroups of fibromyalgia patients. Br. J. Med. Psychol. 71:185-194.

Kvale G, Berg E, Nilsen C et al (1997). Validation of the Dental Fear Scale and the Dental Beliefs Survey in a Norwegian sample. Comm. Dent. Oral Epidemiol. 25:160-164.

Laskin DM (1993). Diagnosis of Pathogy of the temporomandibular joint. Radiolog Clin N Amer 31:135-147.

Lautch H (1971) Dental phobia. Br. J. Psychiat. 119:151-158.

Law A, Logan H and Baron R (1994). Desire for control, felt control, and stress inoculation training during dental treatment. J. Pers. Soc. Psychol. 67:926-936.

Lawson K, Reesor KA, Keefe FJ & Turner JA (1990). Dimensions of pain-related cognitive coping: cross-validation of the factor structure of the Coping Strategy Questionnaire. Pain 43:195-204.

Lazarus R (1975). A cognitively oriented psychologist looks at biofeedback. Amer. Psychol. 30:553-561.

Lefebvre M (1981). Cognitive distortion and cognitive errors in depressed psychiatric and low back pain patients. J Consult. Clin. Psychol 49:517-525.

Lefebvre JC, Lester N & Keefe FJ (1995). Pain in young adults II: The use and perceived effectiveness of pain-coping strategies. Clin. J. Pain 11:36-44.

LeFort S, Gray-Donald K, Rowat K and Jeans ME (1997). Randomized controlled trial of a community-based psychoeducation program for the self-management of chronic pain. Pain 74: 297-306.

Leijdekkers M and Passchier J (1990). Prediction of migraine using psychophysiological and personality variables. Headache 30:445-453.

Leijdekkers M, Passchier J, Goudswaard P, Menges L and Orlebeke J (1990). Migraine patients cognitively impaired? Headache 30:352-358.

Levenson H & Miller J (1976). Multidimensional locus of control in socio-political activists of conservative and liberal ideologies. J Pers. Soc. Psychol. 33:199-208.

Leventhal EA (1984). Aging and the perception of illness. Res Aging 6:119-135.

Leventhal H & Diefenbach M (1991). The active side of illness cognition. In: J.A. Skelton & R.T. Croyle (Eds.) Mental Representation in Health and Illness. Pp. 247-272. New York: Springer Verlag.

Lichstein K, Fischer S, Eakin T, Amberson J, Bertorini T and Hoon P (1991). Psychophysiological parameters of migraine and muscle-contraction headaches. Headache 31:27-34.

Liddell A, di Fazio L, Blackwood J et al (1994). Long-term follow-up of treated dental phobics. Behav. Res. Ther. 32:605-610.

Lindsay S, Humphris G and Barnby G (1987). Exoectations and preferences for routine dentistry in anxious adult patients. Br. Dent. J. 163:120-124.

Lindsay S, Rear S, Wege P and Willard P (1985). Effects of information about discomfort in routine conservation. J. Dent. Res. 64:685.

Lipton J, Ship J and Larach-Robinson D (1993). Estimated prevalence and distribution of reported orofacial pain in the United States. J. Am. Dent. Assoc. 24:115-121.

Lipton R, Stewart W, Celentano D and Reed M (1992). Undiagnosed migraine headaches: A comparison of symptom-based and reported physician diagnosis. Arch. Intern. Med. 152:1273-1278.

List T, Helkimo M & Karlsson R (1993). Pressure pain thresholds in patients with craniomandibular disorders before and after treatment with acupuncture and occlusal splint therapy: a controlled clinical study. J Orofac Pain 7:275-282.

Litaker D, Solomon G and Genzen B (1997). Using pretreatment quality of life perceptions to predict response to sumatriptan in migraineurs. Headache 37:630-634.

Litt M (1996). A model of pain and anxiety associated with acute stressors: distress in dental procedures. Behav. Res. Ther. 34:459-476.

Locker D and Grushka M (1987). The impact of dental and facial pain. J. Dent. Res. 66:1414-1417.

Locker D and Slade G (1988). Prevalence of symptoms associated with temporomandibular disorders in a Canadian population. Comm. Dent. Oral Epidemiol. 16:310-313.

Locker D and Slade G (1993). Oral health and the quality of life among older adults: the oral health impact profile. J Can Dent Assoc 59:830-844.

Logan H, Baron R, Keeley K, Law A, Moreland M and Stein S (1991). Desired and felt control as mediators of stress in a dental setting. Health Psychol. 10:352-359.

Logan H, Baron R and Kohout F (1995). Sensory focus as therapeutic treatments for acute pain. Psychosom. Med. 57:475-484.

Long JH (1992). Occlusal adjustment as treatment for tenderness in the muscles of mastication in category 1 patients. J Pros Dent 67:519-524.

Lorig K, Mazonson P and Holman H (1993). Evidence suggesting that health education for self-management in patients with chronic arthritis has sustained benefits while reducing health care costs. Arth. Rheum. 38:439-446.

Lous I (1978). Treatment of TMJ syndrome by pivots. J Prosthet Dent 40:179-182.

Lumley MA, Asselin LA & Norman S (1997). Alexithymia in chronic pain patients. Compr Psychiat 38:160-165.

Lundh H, Estesson Pl, Rune B & Selvik G (1988). Changes in mandibular position during treatment with disk repositioning onlays: a roentgen stereophotogrammetric study. Oral Surg Oral Med Oral Path 65:657-662.

Lundh H & Westesson PL (1989). Long-term follow-up after occlusal treatment to correct abnormal temporomandibular joint disk position. Oral Surg Oral Med Oral Path 67:2-10.

McAbee G (1998). Malpractice risks associated with prescribing medication for chronic headaches. Headache 38:53-55.

McAdams C, Byrne C & Browne G (1989). Meaning of Illness Questionnaire. Questionnaire and Scoring Manual. McMaster University, School of Nursing.

McCracken LM, Faber SD and Janeck AS (1998). Pain-related anxiety predicts non-specific complaints in persons with chronic pain. Behav. Res. Ther. 36:621-630.

McCracken LM & Gross RT (1993). Does anxiety affect coping with chronic pain? Clin. J. Pain 9:253-9.

McCracken L, Gross R, Sorg P and Edmands T (1993). Prediction of pain in patients with chronic low back pain: Effects of inaccurate prediction and pain-related anxiety. Behav. Res. Ther. 31:647-652.

McCracken L, Zayfert C & Gross R (1992). The Pain Anxiety Symptoms Scale: development and validation of a scale to measure fear of pain. Pain 50:67-73.

McNair D, Lorr M and Doppelman L (1971). Manual for the profile of Mood States. Educational and Industrial Testing Service, San Diego, CA.

McNeil D and Berryman L (1989). Components of dental fear in adults. Behav. Res. Ther. 27:233-236.

Madland G (1988). Preferences for the predictability of discomfort in routine dentistry. Unpublished BSc. thesis, University College London.

Madland G & Feinmann C (1999). Psychiatric perspectives on headache. In: The Effective Management of Headache. Goadsby PJ, Dowson AJ & Miles A (Eds.), UK Key Advances in Clinical Practice Series 2000, Aesculapius Medical Press.

Madland G, Feinmann C & Newman S (2000). Factors associated with anxiety and depression in facial arthromyalgia. Pain, in press.

Magni G (1987). On the relationship between chronic pain and depression when there is no organic lesion. Pain 31:1-21.

Magni G, Moreschi C, Rigatti-Luchini S & Merskey H (1994). Prospective study on the relationship between depressive symptoms and chronic musculoskeletal pain. Pain 56:289-297.

Main CJ (1983). The Modified Somatic Perception Questionnaire (MSPQ). J. Psychosom. Res. 27:503-514.

Main CJ, Parker H, Burton AK, Spanswick CC & Waddell G (1996). Cognitive coping strategies and chronicity in low back pain. Abstracts 8th World Congress on Pain 72.

Maisiak R, Austin J and Heck L (1994). Health status improvement in arthritis patients produced by a telephone counselling intervention: A controlled clinical trial. Arth. Rheum. 37:S289.

Manne SL & Zautra AJ (1990). Couples coping with chronic illness: women with rheumatoid arthritis and their healthy husbands. J. Behav. Med. 13:327-342.

Marbach JJ, Lennon M & Dohrenwend B (1988). Candidate risk factors for temporomandibular pain and dysfunction syndrome: psychosocial, health behavior, physical illness and injury. Pain 34:139-151.

Marbach JJ, Raphael K, Dohrenwend B, Lennon M (1990). The validity of tooth grinding measures: etiology of pain dysfunction syndrome revisited. J Amer Dent Assoc 120:327.

Marbach JJ (1992). The temporomandibular pain dysfunction syndrome personality: fact or fiction? J Oral Rehab 10:545-560.

Marlowe N (1998). Stressful events, appraisal, coping and recurrent headache. J. Clin. Psychol. 54:247-256.

Marteau TM & Bekker H (1992). The development of a six-item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI). Br J Clin Psychol 31:301-306.

Martin P, Nathan P, Milech D and van Keppel M (1989). Cognitive therapy vs. self-management training in the treatment of chronic headaches. Br. J. Clin. Psychol. 28:347-361.

Mason , C, Porter SR, Madland G & Parry J (1997). Early management of dental pain in children and adolescents. J Dent 25:31-34.

Melamed B, Weinstein D, Hawes R and Katin-Borland M (1975). Reduction of fear-related dental management problems with use of filmed modeling. J. Amer. Dent. Ass. 90:822-826.

Melzack R (1987). The short-form McGill Pain Questionnaire. Pain 30:191-197.

Melzack R and Wall PD (1965). Pain mechanisms: A new theory. Science 150:971-979.

Merskey H (1980). Psychological and psychiatric aspects of pain control. In: Smith W, Merskey H & Gross S (Eds.). Pain: Meaning and Management. SP Medical and Scientific Books, NY:105-117.

Merskey H, Albe-Fessard DG, Bonica JJ, Carmen A, Dubner R, Kerr FWL, Lindblom U, Mumford JM, Nathan PW, Noordenbos W, Pagni CA, Renaer MJ, Sternbach RA and Sutherland S (1979). IASP sub-committee on taxonomy. Pain 6:249-252.

Merskey H, Brown J, Brown A, Malhotra L, Morrison D and Ripley C (1985). Psychological normality and abnormality in persistent headache patients. Pain 23:35-47.

Michultka D, Blanchard E, Appelbaum K, Jaccard J and Dentinger M (1989). The refractory headache patient-II. High medication consumption (analgesic rebound) headache. Behav. Res. Ther. 27:411-420.

Miles A, Glover L, Harrison S et al (1995). Chronic facial pain: measurement of depression using the HADS and the BDI. Unpublished.

Milgrom P, Coldwell S, Getz T, Weinstein P and Ramsay D (1997). Four dimensions of fear of dental injections. J. Amer. Dent. Ass. 128:756-762.

Milgrom P, Weinstein P, Kleinknecht R and Getz T (1985). Treating Fearful Dental Patients. Reston, VA.

Miller MC, Murphy R and Buss AH (1981). Consciousness of body: Private and public. J. Pers. Soc. Psychol. 41:397-406.

Mintz SS (1993). Craniomandibular dysfunction in children and adolescents: a review. J Craniomandib Pract 11:224-231.

Mischel W, Zeiss R and Zeiss A (1974). Internal-external control and persistence: validation and implications of the Stanford Preschool Internal-External Scale. J. Pers. Soc. Psychol. 29:265-278.

Mohl ND, Lund JP, Widmer CG & McCall WD Jr (1990). Devices for the diagnosis and treatment of temporomandibular disorders. Part II: Electromyography and sonography. J Pros Dent 63:332-336.

Mohl ND & Dixon DC (1994). Current status of diagnostic procedures for temporomandibular disorders. J Am Dent Assoc 125:56-64.

Mongini F, Defilippi N and Negro C (1997). Chronic daily headache: A clinical and psychological profile before and after treatment. Headache 37:83-87.

Mongini F and Italiano M (2001). TMJ disorders and myogenic facial pain: a discriminative analysis using the McGill Pain Questionnaire. Pain 91: 323-330.

Mongini F, Italiano M, Raviola F and Mossolov A (2000). The MPQ in patients with TMJ pain and with facial pain as a somatoform disorder. J. Craniomandib. Pract. 18:249-256.

Moore R, Abrahamsen R and Bridsgaard I (1996). Hypnosis compared with group therapy and individual desensitization for dental anxiety. Eur. J. Oral Sci. 104:612-618.

Moore R and McQuay H (1997). Single-patient data meta-analysis of 3453 postoperative patients: oral tramadol versus placebo, codeine and combination analgesics. Pain 69:287-94.

Morley S & Pallin V (1995). Scaling the affective domain of pain: a study of the dimensionality of verbal descriptors. Pain 62:39-49.

Morley S, Eccleston C and Williams A (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. Pain 80:1-13.

Morosko T and Simmons F (1966). The effect of audio-analgesia on pain threshold and pain tolerance. J. Dent. Res. 45:1608-1617.

Moss R, Lombardo T, Villarosa G, Cooley J, Simkin L and Hodgson J (1995). Oral habits and TMJ dysfunction in facial pain and non-pain subjects. J. Oral Rehab. 22:79-81.

Murray H, Locker D, Mock D and Tennenbaum H (1996). Pain and the quality of life in patients referred to a craniofacial pain unit. J. Orofac. Pain 10:316-323.

Murray P, Liddell A and Donohue J (1989). A longitudinal study of the contribution of dental experience to dental anxiety in children between 9 and 12 years of age. J. Behav. Med. 12:309-320.

Mumford J, Newton A and Ley P (1973). Personality, pain perception and pain tolerance. Br. J. Psychol. 64:105-107.

National Institutes of Health Technology Assessment Conference Statement. Management of temporomandibular disorders. National Institutes of Health, Washington,DC, 1996.

Nemcovsky CE, Gazit E, Serfati V & Gross M (1992). A comparative study of three therapeutic modalities in a temporomandibular disorder (TMD) population. J Craniomandib Pract 10:148-155.

Newman S, Fitzpatrick R, Revenson T, Skevington S and Williams G. Understanding Rheumatoid Arthritis, Routledge, London, 1996, 237pp.

Nicassio PM, Schoenfeld-Smith K, Radojevic V and Schuman C (1995). Pain coping mechanisms in fibromyalgia: relationship to pain and functional outcomes. J. Rheumatol. 22:1552-1558.

Novy D, Nelson D, Berry L et al (1995). What does the Beck Depression Inventory measure in chronic pain?: a reappraisal. Pain 61:261-270.

Ohno H, Morinushi T, Ohno K & Ogura T (1988). Comparative subjective evaluation and prevalence study of TMJ dysfunction syndrome in Japanese adolescents based on clinical examination. Comm Dent Oral Epidemiol 16:122-126.

Ohrbach R and Dworkin S (1998). Five-year outcomes in TMD: relationship of changes in pain to changes in physical and psychological variables. Pain 74:315-326.

Okeson JP (1987). The effects of hard and soft occlusal splints on nocturnal bruxism. J Amer Dent Assoc 114:788-791.

Okeson JP (1988). Long-term treatment of disk-interference disorders of the temporomandibular joint with anterior repositioning occlusal splints. J Pros Dent 60:611-616.

Okeson JP (1991). Non surgical management of disc-interference disorders. Dent Clin N Amer 35:29-51.

Okeson JP, Kemper JT & Moody PM (1982). A study of the use of occlusion splints in the treatment of acute and chronic patients with craniomandibular disorders. J Pros Dent 48:708-712.

Osman A, Barrios F, Osman J et al (1994). The Pain Anxiety Symptoms Scale: psychometric properties in a community sample. J Beh. Med. 17:511-522.

Parker J, McCrae C, Smarr K, Beck N, Frank R, Anderson S & Walker S (1988). Coping strategies in rheumatoid arthritis. J. Rheumatol. 15:1376-1383.

Parker JC, Smarr KL, Buescher KL, Phillips LR, Frank RG, Beck NC, Anderson SK & Walker SE (1989). Pain control and rational thinking. Arth. Rheum. 32:984-990.

Passchier J, de Boo M, Quaak J and Brienen J (1996). Health-related quality of life of chronic headache patients is predicted by the emotional component of their pain. Headache 36:556-560.

Passchier J, Mourik J, Brienen J and Hunfeld J (1998). Cognitions, emotions, and behavior of patients with migraine when taking medication during an attack. Headache 38:458-464.

Peltola MK, Pernu H, Oikarinen KS and Raustia AM (2000). The effect of surgical treatment of the temporomandibular joint: A survey of seventy patients. Cranio 18: 120-126.

Persson B (1997). Growth environment and personality in adult migraineurs and their migraine-free siblings. Headache 37:159-168.

Peterson RA and Reis S (1992).Anxiety Sensitivity Index Manual (2nd ed.) Worthington, OH: International Diagnostic Systems.

Peveler R (1998). Understanding medically unexplained symptoms: Fatser progress in the next century than in this? J. Psychosom. Res. 45:93-97.

Pfaffenrath V, Diener HC, Isler H, Meyer C, Scholz E, Taneri Z, Wessely P, Zaiser-Kaschel H, Haase W and Fischer W (1993). Effectiveness and tolerance of amitriptyline oxide in chronic tension-type headache - a multicenter double-blind study versus amitiptyline versus placebo. Nervenarzt. 64:114-120.

Pierce CJ & Gale EN (1988). A comparison of different treatments for nocturnal bruxism. J Dent Res 67:597-601.

Pincus T & Madland G (2000). Measuring headache. In: Health Status Measurement in Neurological Disorders. Jenkinson, Fitzpatrick & Jenkinson (Eds.), Radcliffe Medical Press.

Pincus T & Williams A (1999). Models and measurements of depression in chronic pain. J Psychosom Res 47:211-219.

Pollard CA (1984). Preliminary validity study of the Pain Disability Index. Percept. Motor Skills 59:974.

Pollmann L (1993). Sounds produced by the mandibular joint in a sample of healthy workers. J. Orofac. Pain 7:359-361.

Porter SR (1996). Temporomandibular Disorders. In: Porter SR, Scully C (Eds) Innovations and developments of non-surgical management of orofacial disease. Science Reviews, Northwood.

Potter RG & Jones JM (1992). The evolution of chronic pain among patients with musculoskeletal problems: a pilot study in primary care. Br. J. Gen. Practice 42;462-464.

Poulton R, Thomson M, Davies S et al (1997). Good teeth, bad teeth and fear of the dentist. Behav. Res. Ther. 35:327-334.

Prochaska JO and DiClemente CC (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. J. Consult. Clin. Psychol. 51:390-395.

Pullinger AG & Seligman DA (1991a). Overbite and overjet characteristics of refined diagnostic groups of temporomandibular disorder patients. Am J Orthod Dentofac Orthop 100:401-415.

Pullinger AG & Seligman DA (1991b). Trauma history in diagnostic groups of temporomandibular disorders. Oral Surg Oral Med Oral Path 71:529-534.

Pullinger AG, Seligman DA & Solberg WK (1988). Temporomandibular disorders. Part I: Functional status, dentomorphologic features and sex differences in a nonpatient population. J Pros Dent 59:228-235.

Pullinger AG, Seligman DA & Solberg WK (1988). Temporomandibular disorders. Part II: Occlusal factors associated with temporomandibular joint tenderness and dysfunction. J Pros Dent 59:363-367.

Pullinger AG, Seligman DA & Solberg WK (1988). Temporomandibular disorders. Part III: Occlusal and articular factors associated with muscle tenderness. J Pros Dent 59:483-489.

Quayle AA, Gray RHM, Metcalfe RJ, Guthrie E & Wastell D (1990). Soft occlusal splint therapy in the treatment of migraine and other headaches. J Dent 18:123-129.

Radloff L (1977). The CES-D scale: a self-report depression scale for use in the general population. Appl. Psychol. Meas. 1:385-401.

Rapaport A (1987). Characteristics and Treatment of Analgesic Rebound Headache. Springer, New York.

Raphael KG and Marbach JJ (1997). Evidence-based care of musculoskeletal facial pain: implications for the clinical science of dentistry. J. Amer. Dent. Ass. 28:73-79.

Raphael KG and Marbach JJ (1997). When did your pain start?: Reliability of self-reported age of onset of facial pain. Clin. J. Pain 13:352-359.

Rasmussen BK, Jensen R and Olesen J (1992). Impact of headache on sickness absence and utilisation of medical services: A Danish population study. J. Epidemiol. Comm. Health 46:443-446.

Rasmussen BK and Olesen J (1994). Epidemiology of migraine and tension-type headache. Curr. Opinion Neurol. 7:264-271.

Regan CA, Lorig K & Thoresen CE (1988). Arthritis appraisal and ways of coping: scale development. Arth. Care Res. 3:139-150.

Reisine S and Weber J (1989). The effects of temporomandibular joint disorders on patients' quality of life. Comm. Dent. Health 6:257-270.

Revenson TA & Felton BJ (1989). Disability and coping as predictors of psychological adjustment to rheumatoid arthritis. J. Consult. Clin. Psychol. 57:344-348.

Reynolds DJ & Hovanitz CA (2000). Life event stress and headache frequency revisited. Headache 40:111-118.

Riley JL, Robinson ME and Geissner ME (1999). Empirical subgroups of the Coping Strategies Questionniare - Revised: A multisample study. Clin. J. Pain 15:111-116.

Roberts CA, Tallents RH, Katzberg RW et al (1987). Comparison of internal derangements of the TMJ with occlusal findings. Oral Surg Oral Med Oral Path 63:645-650.

Rodin G, Craven J & Littlefield C (1991). Depression in the mentally ill: an integrated approach. Brunner/Mazel, NY.

Rokke PD & al'Absi M (1992). Matching pain coping strategies to the individual: a prospective validation of the cognitive coping strategy inventory. J. Behav. Med. 15:611-625.

Romanelli GG, Harper R, Mock D, Pharoah MJ & Tenenbaum HC (1993). Evaluation of temporomandibular joint internal derangement. J Orofac Pain 7:254-262.

Romano J & Turner J (1985). Chronic pain and depression: does the evidence support a relationship? Psychol. Bull. 97:18-34.

Rosenstiel A and Keefe F (1983). The use of coping strategies in chronic low back patients: relationship to patient characteristics and current adjustment. Pain 17: 33-44.

Rotter J (1966). Generalized expectancies for internal versus external control of reinforcement. Psychol. Monogr. Gen. Appl. 80: no.609.

Rudy T, Kerns R & Turk D (1988). Chronic pain and depression: towards a cognitive-behavioral mediation model. Pain 35:129-140.

Saadah H (1997). Headache fear. J. Okla. State Med. Ass. 90:179-184.

Sadowsky C (1992). The risk of orthodontic treatment for producing temporomandibular mandibular disorders: a literature overview. Am J Orthod Dentofac Orthop 101:79-83.

Salonen L, Hellden L & Carlsson GE (1990). Prevalence of signs and symptoms of dysfunction in the masticatory system: An epidemiologic study in an adult Swedish population. J Craniomandib Disord Fac Oral Pain 4:241-250.

Sano T & Westesson PL (1995). Magnetic resonance imaging of the temporomandibular joint. Increased T2 signal in the retrodiskal tissue of painful joints. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 79:511-516.

Schiffman E, Fricton J and Haley D (1992). The relationship of occlusion, parafunctional habits and recent life events to mandibular dysfunction in a non-patient population. J. Oral Rehab. 19:201-223.

Schiffman EL, Friction JR, Haley DP & Shapiro BL (1990). The prevalence and treatment needs of subjects with temporomandibular disorders. J Amer Dent Assoc 120:295-303.

Schmitz U, Saile H and Nilges P (1996). Coping with chronic pain: flexible goal adjustment as an interactive buffer against pain-related distress. Pain 67:41-51.

Schnurr R, Brooke R and Rollman G (1990). Psychosocial correlates of temporomandibular joint pain and dysfunction. Pain 42:153-165.

Schnurr R, Rollman G and Brooke R (1991). Are there psychologic predictors of treatment outcome in temporomandibular joint pain and dysfunction? Oral Surg. Oral Med. Oral Pathol. 72:550-558.

Schulte JK, Anderson GC, Hathaway KM & Will ET (1993). Psychometric profiles and related pain characteristics of temporomandibular disorder patients. J Orofac Pain 7:247-253.

Schuurs A, Duivenvoorden H, Makkes P et al (1988). Personality traits of patients suffering extreme dental anxiety. Comm. Dent. Oral Epidemiol. 16:38-41.

Schwartz SM and Gramling SE (1997). Cognitive factors associated with facial pain. Cranio 15: 261-266.

Scott D, Hirscman R and Schroder K (1984). Historical antecedents of dental anxiety. J. Amer. Dent. Ass. 108:42-45.

Selby A (1985). Physiotherapy in the management of temporomandibular disorders. Aust Dent J 30:273-280.

Seligman DA & Pullinger AG (1989). Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Disord Fac Oral Pain 3:227-236.

Seligman D, Pullinger A (1991). The role of functional occlusal relationships in temporomandibular disorders: A review. J. Craniomandib. Disord. Facial Oral Pain 5:265-279.

Seligman D, Pullinger A and Solberg W (1988). The prevalence of dental attrition and its association with factors of age, gender, occlusion and TMJ symptomatology. J. Dent. Res. 67:1323-1333.

Shaw A and Niven N (1996). Theoretical concepts and practical applications of hypnosis in the treatment of children and adolescents with dental fear and anxiety. Br. Dent. J. 180:11-16.

Sheikholeslam A, Holmgren K and Riise C (1986). A clinical and electromyographic study of the long-term effects of an occlusal splint on the temporal and masseter muscles in patients with functional disorders and nocturnal bruxism. J. Oral Rehabil. 13:137-145.

Shan SC & Yun WH (1991). Influence of an occlusal splint on integrated electromyography of the masseter muscles. J Oral Rehab 18:253-256.

Siegel L and Peterson L (1980). Stress reduction in young dental patients through coping skills and sensory information. J. Consult.Clin. Psychol. 6:785-787.

Siegel L and Peterson L (1981). Maintenance effects of coping skills and sensory information on young children's response to repeated dental procedures. Behav. Ther. 12:530-535.

Sigurdsson A and Maixner W (1994). Effects of experimental and clinical noxious counterirritants on pain perception. Pain 57:265-75.

Sipila K, Veijola J, Jokelainen J, Jarvelin MR, Oikarinen KS, Raustia AM and Joukamaa M (2001). Association of symptoms of TMD and orofacial pain with alexithymia: an epidemiological study of the Northern Finland 1966 Birth Cohort. Cranio 19:246-251.

Skelton JA & Croyle RT (1991). Mental Representation in Health and Illness (Eds.). New York: Springer Verlag.

Skinner H (1982). The drug abuse screening test. Addict. Behav. 7:363-371.

Slade G and Spencer J (1994). Development and evaluation of the Oral Health Impact Profile. Comm. Dent. Health 11:3-11.

Slater M, Hall H, Atkinson J et al (1991). Pain and impairment beliefs in chronic low back pain: validation of the Pain and Impairment Relationship Scale (PAIRS). Pain 44:51-56.

Smith T, O'Keefe J & Christensen A (1994). Cognitive distortion and depression in chronic pain: association with diagnosed disorders. J Consult. Clin. Psychol. 92:195-198.

Snow-Turek AL, Norris MP & Tan G (1996). Active and passive coping strategies in chronic pain patients. Pain 64:455-462.

Solberg WK, Clark GT & Rugh JD (1975). Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. J Oral Rehab 2:215-223.

Solomon G (1997). Evolution of the measurement of quality of life in migraine. Neurology 48 Supp.3:10-15.

Speculand B, Goss AN, Hughes A, Spence ND & Pilowsky I (1983). Temporomandibular joint dysfunction: Pain and illness behaviour. Pain 17:139-150.

Spielberger CD (1988). State-Trait Anger Expression Inventory, Revised Research Edition: Professional Manual. Psychological Assessment Resources, Inc: Odessa, FL.

Spielberger CD, Edwards C, Lushene R, Montuori J and Platzek D (1973). State-Trait Anxiety Inventory for Children. Consulting Psychologists Press, Palo Alto, CA.

Spielberger CD, Gorsuch RL and Lushene RE (1970). Manual for the State-Trait Anxiety Inventory. Consulting Psychologist Press, Palo Alto, CA.

Spielberger CD, Gorsuch R, Lushene R, Vagg P and Jacobs G (1983). Manual for the State-Trait Anxiety Inventory (Form Y). Consulting Psychologists Press, Palo Alto, CA.

Spielberger CD, Johnson EH, Russell SF, Crane RJ, Jacobs GA and Worden TJ (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In Chesney MA and Rosenmann RH (Eds.): Anger and Hostility in Cardiovascular and Behavioral Disorders. Washington, DC: Hemisphere Publishing.

Spierings E, Sorbi M, Maassen G and Honkoop P (1997). Psychophysical precedents of migraine in relation to the time of onset of the headache: The migraine time line. Headache 37:217-220.

Spinhoven P, ter Kuile MM, Linsen ACG & Gazendam B (1989). Pain coping strategies in a Dutch population of chronic low back pain patients. Pain 37:77-83.

Spitzer R, Endicott J & Robins E (1978): Research diagnostic criteria: rationale and reliability. Arch. Gen. Psychiat. 35:773-782.

Steblay N and Beaman A (1982). Reduction of fear during dental treatment through reattribution techiques. J. Amer. Dent. Ass. 105:1006-1009.

Stein S, Loft G, Davis H & Hart DL (1982). Symptoms of TMJ dysfunction as related to stress measured by the social readjustment rating scale. J Pros Dent 47:545.

Sternbach R (1986). Pain and 'hassles' in the United States: findings of the Nuprin Pain Report. Pain 27:69-80.

Stronks D, Tulen J, Verheij R, Boomsma F, Fekkes D, Pepplinkhuizen L, Mantel G and Passchier J (1998). Serotonergic, catecholaminergic, and cardiovascular reactions to mental stress in female migraine patients. A controlled study. Headache 38:270-280.

Sullivan MJL, Bishop S and Pivik J (1995). The Pain Catastophizing Scale: Development and valaidation. Psychol. Assess. 7:524-532.

Sullivan M and D'Eon J (1990). Relation between catastrophizing and depression in chronic pain patients. J. Abnorm. Psychol. 99:260-263.

Sullivan MJL and Neish N (1999). The effects of disclosure on pain during dental hygiene treatment: The moderating role of catastrophizing. Pain 79:155-163.

Sullivan M, Reesor K, Mikail S et al (1992). The treatment of depression in chronic low back pain: review and recommendations. Pain 50:5-13.

Suls J and Wan C (1989). Effects of sensory and procedural information on coping with stressful medical procedures and pain: a meta-analysis. J. Consult. Clin. Psychol. 57:372-379.

Sundblom D, Haikonen S, Niemi-Pynttari J and Tigerstedt I (1994). Effect of spiritual healing on chronic idiopathic pain: a medical and psychological study. Clin. J. Pain 10:296-302.

Swartzman LC, Gwadry FG, Shapiro AP & Teasell RW (1994). The factor structure of the Coping Strategies Questionnaire. Pain 57:311-316.

Szentpetery A (1993). Clinical utility of mandibular movement ranges. J. Orofac. Pain 7:163-168.

Tallents RH, Catania J & Sommers E (1986). Temporomandibular joint findings in pediatric populations and young adults: a critical review. Angle Orthodont 61:7-13.

Tallents RH, Katzberg RW, Macher DJ & Roberts CA (1990). Use of protrusive splint therapy in anterior disk displacement of the temporomandibular joint : A 1- to 3-year follow-up. J Pros Dent 63:336-341.

Tellegen A (1976). Differential Personality Questionnaire. University of Minnesota, Minneapolis, MN.

Tellegen A and Atkinson G (1974). Openness to absorbing and self-altering experiences ("absorption"), a trait related to hypnotic susceptibility. J. Abnorm. Psychol. 83:268-277.

ter Kuile M, Spinhoven P and Linssen A (1995). Responders and nonresponders to autogenic training and cognitive self-hypnosis: prediction of short- and long-term success in tension-type headache patients. Headache 35:630-636.

ter Kuile M, Spinhoven P, Linssen A and van Houwelingen H (1995). Cognitive coping and appraisal processes in the treatment of chronic headaches. Pain 64:257-264.

Toomey T, Mann J, Abashian S & Thompson-Pope S (1991). Relationship between perceived self- control of pain, pain description and functioning. Pain 45:129-133.

Truelove DL, Sommers EE, LeResche L et al (1992). Clinical diagnostic criteria for TMD: new classification permits multiple diagnoses. J Amer Dent Assoc 123:47-56.

Tsukiyama Y, Baba K and Clark GT (2001). An evidence-based assessment of occlusal adjustment as a treatment for temporomandibular disorders. J Prosthet. Dent. 86:57-66.

Turk D, Okifuji A & Scharff L (1995). Chronic pain and depression: role of perceived impact and perceived control in different age cohorts. Pain 61:93-101.

Turk D & Rudy T (1992). Cognitive factors and persistent pain: a glimpse into Pandora's box. Cog. Ther. Res. 16:99-122.

Turk DC, Rudy TE, Kubinski JA, Zaki HS & Greco CM (1996). Dysfunctional patients with temporomandibular disorders: evaluating the efficacy of a tailored treatment protocol. J Consult Clin Psychol 64:139-146.

Turner JA & Clancy S (1986). Strategies for coping with chronic low back pain: relationships to pain and disability. Pain 24: 355-364.

Turner JA, Clancy S & Vitaliano PP (1987). Relationships of stress, apraisal and coping to chronic low back pain. Behav. Res. Ther. 25: 281-288.

Turner JA, Dworkin SF, Mancl L, Huggins KH and Truelove EL (2001). The roles of beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorders. Pain 92: 41-51.

Turner JA, Jensen MP & Romano JM (2000). Do beliefs, coping, and catastrophizing independently predict functioning in patients with chronic pain? Pain 85:115-125.

Turner J, Whitney C, Dworkin S, Massoth D and Wilson L (1995). Do changes in patient beliefs and coping strategies predict temporomandibular disorder treatment outcomes? Clin. J. Pain 11:177-188.

Turp J, Kowalski C and Stohler C (1997). Pain descriptors characteristic of persistent facial pain. J. Orofac. Pain 11:285-290.

Tversky J, Reade PC, Gerschman JA et al (1991). Role of depressive illness in the outcome of treatment of temporomandibular joint pain-dysfunction syndrome. Oral Surg Oral Med Oral Path 71:696-699.

Ulrich V, Russell MB, Jensen R and Olesen J (1996). A comparison of tension-type headache in migraineurs and in non-migraineurs: A population study. Pain 67:501-506.

Ursin H, Endersen IM and Ursin G (1988). Psychological factors and self-reports of muscle pain. Eur. J. Appl. Psychol. 57:282-290.

Vallerand W, Vallerand A and Heft M (1994). The effects of postoperative preparatory information on the clinical course following third molar extraction. J. Oral Maxillofac. Surg. 52:1165-1170.

Vassend O (1992). Anxiety, pain and discomfort associated with dental treatment. Behav. Res. Ther. 31:659-666.

Venham L, Bengston D and Cipes M (1977). Children's response to sequential dental visits. J. Dent. Res. 56:454-459.

Verri A, Proietti Cecchini A, Galli C, Granella F, Sandrini G and Nappi G (1998). Psychiatric comorbidity in chronic daily headache. Cephalalgia 18 Supp.21:45-49.

Vimpari SS, Knuuttila ML, Sakki TK & Kivela SL (1995). Depressive symptoms associated with symptoms of the temporomandibular joint pain and dysfunction syndrome. Psychosom Med 57:439-444.

Vitaliano PP, Russo J, Carr JE, Maiuro RD & Becker J (1985). The Ways of Coping Checklist: revision and psychometric properties. Mult. Behav. Res. 20:3-26.

von Korff M and Simon G (1996). The relationship between pain and depression. Br. J. Psychiat. 168:S101-108.

Voudouris N, Peck C and Coleman G (1990). The role of conditioning and verbal expectancy in the placebo response. Pain 43:121-128.

Wade J, Dougherty L, Hart R and Cook D (1992). Patterns of normal personality structure among chronic pain patients. Pain 48:37-43.

Wagner T, Patrick D, Galer B and Berzon R (1996). A new instrument to assess the long-term quality of life effects from migraine: Development and psychometric testing of the MSQOL. Headache 36:484-492.

Walker L and Zeman J (1992). Parental responses to child illness behavior. J. Pediatr. Psychol. 17:49-71.

Wall PD (1994). The placebo and the placebo response. In: Textbook of Pain, 3rd ed. Wall PD and Melzack R (eds.). Churchill Livingstone, London.

Wall PD (1999). Pain: The Science of Suffering. Weidenfeld & Nicolson, London.

Wallston F, Wallston B & DeVellis R (1978). Development of the Multidimensional Health Locus of Control (MHLC) scales. Health Educ. Monogr. 6:160-170.

Wallston B, Wallston K, Kaplan G and Maides S (1976). development and validation of the Health Locus of Control (HLC) Scale. J. Consult. Clin. Psychol. 44:580-585.

Wang W, Wang Y-H, Fu X-M, Sun Z-M and Schoenen J (1999). Auditory evoked potentials and multiple personality measures in migraine and post-traumatic headaches. Pain 79:235-242.

Wardle J (1983). Psychological management of anxiety and pain during dental treatment. J. Psychosom. Res. 27:399-402.

Watson D, Clark L and Tellegen A (1988). Development and validation of brief measures of positive and negative affect: The PANAS scale. J. Pers. Soc. Psychol. 54:1063-1070.

Weir R, Browne G, Roberts J et al (1994). The Meaning of Illness Questionnaire: further evidence for its reliability and validity. Pain 58:377-386.

Weisenberg M, Kreindler M, Schachat R and Werboff J (1975). Pain: anxiety and attitudes in black, white and Puerto Rican patients. Psychosom. Med. 37:123-135.

Weitzenhoffer A and Hilgard E (1959). Standard Hypnotic Susceptibility Scale. Consulting Psychologists Press, Palo Alto, CA.

Wesley A, Gatchel R, Polatin P et al (1991). Differentiation between somatic and cognitive-afferent components in commonly used measures of depression in patients with chronic low-back pain. Spine 16: S213-215.

White CS & Dolwick MF (1992). Prevalence and variance of temporomandibular dysfunction in orthognathic surgery patients. Int J Adult Orthod Orthognath Surg 7:7-14.

Widerstrom E, Aslund P, Gustafsson L-E, Mannheimer C, Carlsson S and Andersson S (1992). Relations between experimentally induced tooth pain threshold

changes, psychometrics and clinical pain relief following TENS. A retrospective study in patients with long-lasting pain. Pain 51:281-287.

Widerstrom-Noga E, Dyrehag L-E, Borglum-Jensen O, Aslund P, Wenneberg B and Andersson S (1998). Pain threshold response to two different modes of sensory stimulation in patients with orofacial muscular pain: psychologic considerations. J. Orofac. Pain 12:27-34.

Wilkie D, Savedra M, Holzemer W, Tesler M and Paul S (1990). Use of the McGill Pain Questionnaire to measure pain: A meta-analysis. Nurs. Res. 39:36-41.

Wilkinson T, Hansson TL, McNeill C & Marcell T (1992). A comparison of the success of 24 hours occlusal splint therapy versus nocturnal occlusal splint therapy in reducing craniomandibular disorders. J Craniomandib Disord Fac Oral Pain 6:64-70.

Williams A & Richardson P (1993). What does the BDI measure in chronic pain? Pain 55:259-266.

Williams D & Keefe F (1991). Pain beliefs and the use of cognitive-behavioral coping strategies. Pain 46:185-190.

Williams D, Robinson M & Geisser M (1994). Pain beliefs: assessment and utility. Pain 59:71-78.

Williams D & Thorn B (1989). An empirical assessment of pain beliefs. Pain 36:351-358

Yuasa H, Kurita K; Treatment Group on Temporomandibular Disorders (2001). Randomized clinical trial of primary treatment for temporomandibular joint disk displacement without reduction and without osseous changes: a combination of NSAIDs and mouth-opening exercises versus no treatment. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 91:671-675.

Zautra A, Marbach JJ, Raphael K, Dohrenweld B, Lennon MC & Kenny D (1995). The examination of myofascial pain and its relationship to psychological distress among women. Health Psychol. 4:223-31.

Ziegler D and Paolo A (1996). Self-reported disability due to headache: A comparison of clinic patients and controls. Headache 36:476-480.

Zigmond A and Snaith R (1983). The Hospital Anxiety and Depression Scale. Acta Psychiatr. Scand. 67:361-367.

Zung W (1965). A self-rating depression scale. Arch. Gen. Psychiat. 12:63-70.

Appendix I.

(Patient Information Letter)

CONFIDENTIAL

November 1997

Dear

You are currently on a waiting list for consultation regarding facial pain. In the meantime, I wonder if you might be interested in taking part in the following research project:

A Study of Beliefs and Coping in Patients with Facial Pain

This study aims to investigate the relationships between what we believe about the nature and consequences of pain, and how we cope with pain, and the degree to which facial pain affects our lives. Previous research has shown that these factors influence how well people are able to adjust to their pain.
The results of the study will be used to produce a treatment programme for facial pain sufferers, which will aim to alter pain-beliefs and ways of coping in order to improve adjustment and prevent the development of long-term problems.

You are invited to attend for a 30 minute appointment to complete a questionnaire and to have your face and jaws examined.
You will be asked about your general health and any medicines which you are taking.
Any information you give will be treated with complete confidentiality.

You are under no obligation to take part in this study but your participation will be gratefully appreciated and extremely helpful. If you decide to take part, you may withdraw at any time without having to give a reason. Your decision whether or not to take part will not affect your treatment in any way.

If you are interested and able to participate, please contact the department.

Yours sincerely,

Geir Madland
Research Fellow

Consent Form

Please read this form carefully. Please ask the researcher if you do not understand or would like more information.

A Study of Beliefs and Coping in Patients with Facial Pain

This study aims to investigate the relationships between what we believe about the nature and consequences of pain, and how we cope with pain, and the degree to which facial pain affects our lives. Previous research has shown that these factors influence how well patients are able to adjust to their pain.
The results of the study will be used to produce a treatment programme for facial pain sufferers, which will aim to alter pain-beliefs and ways of coping in order to improve patient adjustment and prevent the development of long-term problems.

You are invited to complete a questionnaire, which will take approximately 20 minutes.
You will be asked about your general health and any medicines which you are taking.
Any information you give will be treated with complete confidentiality.
If you suffer from persistent joint and muscle pain in the face, your face and jaws will be examined by the investigator.

You are under no obligation to take part in this study but your participation will be gratefully appreciated and extremely helpful. If you decide to take part, you may withdraw at any time without having to give a reason. Your decision, whether to take part or not, will not affect your care and management in any way.

Please complete the following statement:

I, ..., understand the purpose and nature of this research project, the details of which have been explained to me, and I agree to participate.

Signature ..... Date ...../...../.....

I have explained the purpose and nature of this research to the above patient, and emphasised that his/her participation is voluntary and confidential.

Signature ..... Date ...../...../.....

The Questionnaire

Thank you for agreeing to take part in this study.

Please follow the instructions for each section and answer **all** the questions.

Your answers will be treated with complete confidentiality.

The questionnaire takes approximately 20 minutes to complete.

Please give the following information about yourself:

age: _____ years

sex: male / female

are you: single / living with a partner / married / separated / divorced / widowed ?

occupation: _____

number of years in full-time education: _____

Now please continue.....

We are interested in what you think about **pain in general.**
For each item please indicate your opinion by **underlining** one of the following words in each sentence:

always / almost always / often / sometimes / rarely / never

There are no right or wrong answers: it is important that you respond according to your actual beliefs, not according to how you feel you should believe or how you think we want you to believe.
Please make sure that you answer **ALL** the questions.

1. Pain is **always / almost always / often / sometimes / rarely / never** the result of damage to the tissues of the body.

2. Physical exercise **always / almost always / often / sometimes / rarely / never** makes pain worse.

3. It is **always / almost always / often / sometimes / rarely / never** impossible to do much for oneself to relieve pain.

4. Being anxious **always / almost always / often / sometimes / rarely / never** makes pain seem worse.

5. Experiencing pain is **always / almost always / often / sometimes / rarely / never** a sign that something is wrong with the body.

6. Being in pain **always / almost always / often / sometimes / rarely / never** prevents you from enjoying hobbies and social activities.

7. When relaxed, pain is **always / almost always / often / sometimes / rarely / never** easier to cope with.

8. The amount of pain is **always / almost always / often / sometimes / rarely / never** related to the amount of damage.

9. Thinking about pain **always / almost always / often / sometimes / rarely / never** makes it worse.

10. It is **always / almost always / often / sometimes / rarely / never** impossible to control pain on your own.

11. Pain is **always / almost always / often / sometimes / rarely / never** a sign of illness.

12. Feeling depressed **always / almost always / often / sometimes / rarely / never** makes pain seem worse.

We are interested in how you respond to being in **pain**.
Please indicate how often you do each of the following, **in response to your pain**, by **circling** the appropriate number:

1. I try to feel distant from the pain, almost as if the pain was in somebody else's body.
not at all 0 1 2 3 4 5 6 very often

2. I leave the house and do something, such as going to the cinema or shopping.
not at all 0 1 2 3 4 5 6 very often

3. I try to do something pleasant.
not at all 0 1 2 3 4 5 6 very often

4. I don't think of it as pain but rather as a dull or warm feeling.
not at all 0 1 2 3 4 5 6 very often

5. I read.
not at all 0 1 2 3 4 5 6 very often

6. I tell myself to be brave and carry on despite the pain.
not at all 0 1 2 3 4 5 6 very often

7. I tell myself that I can overcome the pain.
not at all 0 1 2 3 4 5 6 very often

8. I count numbers in my head or run a song through my mind.
not at all 0 1 2 3 4 5 6 very often

9. I just think of it as some other sensation, such as numbness.
not at all 0 1 2 3 4 5 6 very often

10. I play mental games with myself to keep my mind off the pain.
not at all 0 1 2 3 4 5 6 very often

11. I try not to think of it as my body, but rather as something separate from me.
not at all 0 1 2 3 4 5 6 very often

12. I don't think about the pain.
not at all 0 1 2 3 4 5 6 very often

13. I tell myself it doesn't hurt.
not at all 0 1 2 3 4 5 6 very often

14. I tell myself I can't let the pain stand in the way of what I have to do.
not at all 0 1 2 3 4 5 6 very often

Please answer specifically about your pain.

15. I don't pay any attention to the pain.
not at all 0 1 2 3 4 5 6 very often

16. No matter how bad it gets, I know I can handle it.
not at all 0 1 2 3 4 5 6 very often

17. I pretend it's not there.
not at all 0 1 2 3 4 5 6 very often

18. I replay in my mind pleasant experiences in the past.
not at all 0 1 2 3 4 5 6 very often

19. I think of people I enjoy doing things with.
not at all 0 1 2 3 4 5 6 very often

20. I imagine that the pain is outside of my body.
not at all 0 1 2 3 4 5 6 very often

21. I just go on as if nothing happened.
not at all 0 1 2 3 4 5 6 very often

22. I see it as a challenge and don't let it bother me.
not at all 0 1 2 3 4 5 6 very often

23. Although it hurts, I just keep on going.
not at all 0 1 2 3 4 5 6 very often

24. I try to be around other people.
not at all 0 1 2 3 4 5 6 very often

25. I ignore it.
not at all 0 1 2 3 4 5 6 very often

26. I think of things I enjoy doing.
not at all 0 1 2 3 4 5 6 very often

27. I do anything to get my mind off the pain.
not at all 0 1 2 3 4 5 6 very often

28. I do something I enjoy, such as watching TV or listening to music.
not at all 0 1 2 3 4 5 6 very often

29. I pretend it's not a part of me.
not at all 0 1 2 3 4 5 6 very often

Please answer specifically about your pain.

30. I do something active, like household chores or projects.
not at all 0 1 2 3 4 5 6 very often

31. It's terrible and I feel it's never going to get any better.
not at all 0 1 2 3 4 5 6 very often

32. It's awful and I feel that it overwhelms me.
not at all 0 1 2 3 4 5 6 very often

33. I feel my life isn't worth living.
not at all 0 1 2 3 4 5 6 very often

34. I know somebody will be here to help me and it will go away for a while.
not at all 0 1 2 3 4 5 6 very often

35. I pray to God it won't last long.
not at all 0 1 2 3 4 5 6 very often

36. I try to think years ahead, what everything will be like after I've gotten rid of the pain.
not at all 0 1 2 3 4 5 6 very often

37. I have faith in doctors that someday there will be a cure for my pain.
not at all 0 1 2 3 4 5 6 very often

38. I worry all the time about whether it will end.
not at all 0 1 2 3 4 5 6 very often

39. I pray for the pain to stop.
not at all 0 1 2 3 4 5 6 very often

40. I feel I can't stand it anymore.
not at all 0 1 2 3 4 5 6 very often

41. I rely on my faith in God.
not at all 0 1 2 3 4 5 6 very often

42. I feel like I can't go on.
not at all 0 1 2 3 4 5 6 very often

43. I feel I have control over the pain.
no control 0 1 2 3 4 5 6 complete control

44. I am able to decrease the pain.
not at all 0 1 2 3 4 5 6 completely

Please answer this section with reference to your **jaw / face pain** over the past few days.
If a word **does not** describe your pain, tick **'NONE'** next to that word.
If a word **does** describe your pain, indicate whether you experience this sensation to a **'MILD'**, **'MODERATE'** or **'SEVERE'** degree, by ticking next to the relevant word.
Please make sure you place a tick in one of the categories for each word.

	NONE	MILD	MODERATE	SEVERE
THROBBING	———	———	———	———
SHOOTING	———	———	———	———
STABBING	———	———	———	———
SHARP	———	———	———	———
CRAMPING	———	———	———	———
GNAWING	———	———	———	———
HOT/BURNING	———	———	———	———
ACHING	———	———	———	———
HEAVY	———	———	———	———
TENDER	———	———	———	———
SPLITTING	———	———	———	———
TIRING/EXHAUSTING	———	———	———	———
SICKENING	———	———	———	———
FEARFUL	———	———	———	———
PUNISHING/CRUEL		———		———

Please answer this section with reference to the pain you have **right at this moment**.

a) Draw a line through the scale below to indicate where your pain is at the moment (e.g. ----/----), imagining that the line indicates the ladder going from no pain to the worst possible pain.

NO PAIN ——————————————————— **WORST POSSIBLE PAIN**

b) Tick next to **one** of the following words / groups of words to indicate how intense your pain is **at the moment**.

NO PAIN .....
MILD .....
DISCOMFORTING .....
DISTRESSING .....
HORRIBLE .....
EXCRUCIATING .....

Read each item and place a firm tick in the box opposite the reply which comes closest to how you have been feeling **in the past few weeks**. Don't take too long over your replies, we want your immediate reaction. **Tick only one box in each section.**

I feel tense or 'wound up':
- Most of the time [ ]
- A lot of the time [ ]
- Time to time, occasionally [ ]
- Not at all [ ]

I feel as if I am slowed down:
- Nearly all the time [ ]
- Very often [ ]
- Sometimes [ ]
- Not at all [ ]

I still enjoy the things I used to enjoy:
- Definitely as much [ ]
- Not quite as much [ ]
- Only a little [ ]
- Hardly at all [ ]

I get a sort of frightened feeling like 'butterflies' in my stomach:
- Not at all [ ]
- Occasionally [ ]
- Quite often [ ]
- Very often [ ]

I get a sort of frightened feeling as if something awful is about to happen:
- Very definitely [ ]
- Yes, but not too badly [ ]
- A little, it doesn't worry me [ ]
- Not at all [ ]

I have lost interest in my appearance:
- Definitely [ ]
- I don't take as much care as I should [ ]
- I may not take quite as much care [ ]
- I take just as much care as ever [ ]

I can laugh and see the funny side of things:
- As much as I always could [ ]
- Not quite so much now [ ]
- Definitely not so much now [ ]
- Not at all [ ]

I feel restless as if I have to be on the move:
- Very much indeed [ ]
- Quite a lot [ ]
- Not very much [ ]
- Not at all [ ]

Worrying thoughts go through my mind:
- A great deal of the time [ ]
- A lot of the time [ ]
- From time to time, not too often [ ]
- Only occasionally [ ]

I look forward with enjoyment to things:
- As much as I ever did [ ]
- Rather less than I used to [ ]
- Definitely less than I used to [ ]
- Hardly at all [ ]

I feel cheerful:
- Not at all [ ]
- Not often [ ]
- Sometimes [ ]
- Most of the time [ ]

I get sudden feelings of panic:
- Very often indeed [ ]
- Quite often [ ]
- Not very often [ ]
- Not at all [ ]

I can sit at ease and feel relaxed:
- Definitely [ ]
- Usually [ ]
- Not often [ ]
- Not at all [ ]

I can enjoy a good book or the TV:
- Often [ ]
- Sometimes [ ]
- Not often [ ]
- Very seldom [ ]

We are interested in how **problems with your jaw or face** affect your life. Please answer each of the following questions by **circling** one number for each, using the response choices listed below.

0 = never **1** = hardly ever **2** = sometimes **3** = fairly often **4** = very often

---

1. Have you had difficulty chewing any foods because of problems with your jaw or face? **0 1 2 3 4**

2. Have you had trouble pronouncing any words because of problems with your jaw or face? **0 1 2 3 4**

3. Have you noticed a tooth which doesn't look right? **0 1 2 3 4**

4. Have you felt that your appearance has been affected because of problems with your jaw or face? **0 1 2 3 4**

5. Have you felt that your breath has been stale because of problems with your jaw or face? **0 1 2 3 4**

6. Have you felt that your sense of taste has worsened because of problems with your jaw or face? **0 1 2 3 4**

7. Have you had food catching in your teeth? **0 1 2 3 4**

8. Have you felt that your digestion has worsened because of problems with your jaw or face? **0 1 2 3 4**

9. Have you had painful aching in your mouth? **0 1 2 3 4**

10. Have you had a sore jaw? **0 1 2 3 4**

11. Have you had headaches because of problems with your jaw or face? **0 1 2 3 4**

12. Have you had sensitive teeth, for example, due to hot or cold foods or drinks? **0 1 2 3 4**

13. Have you had toothache? **0 1 2 3 4**

14. Have you had painful gums? **0 1 2 3 4**

15. Have you found it uncomfortable to eat any foods because of problems with your jaw or face? **0 1 2 3 4**

16. Have you had sore spots in your mouth? **0 1 2 3 4**

0 = never **1** = hardly ever **2** = sometimes **3** = fairly often **4** = very often

---

17. Have you been worried because of problems with your jaw or face?
0 1 2 3 4

18. Have you been self-conscious because of problems with your jaw or face?
0 1 2 3 4

19. Have you been miserable because of problems with your jaw or face?
0 1 2 3 4

20. Have you felt uncomfortable about the appearance of your jaw or face?
0 1 2 3 4

21. Have you felt tense because of problems with your jaw or face?
0 1 2 3 4

22. Has your speech been unclear because of problems with your jaw or face?
0 1 2 3 4

23. Have people misunderstood some of your words because of problems with your jaw or face?
0 1 2 3 4

24. Have you felt that there has been less flavour in your food because of problems with your jaw or face?
0 1 2 3 4

25. Have you been unable to brush your teeth properly because of problems with your jaw or face?
0 1 2 3 4

26. Have you had to avoid eating some foods because of problems with your jaw or face?
0 1 2 3 4

27. Has your diet been unsatisfactory because of problems with your jaw or face?
0 1 2 3 4

28. Have you avoided smiling because of problems with your jaw or face?
0 1 2 3 4

29. Have you had to interrupt meals because of problems with your jaw or face?
0 1 2 3 4

30. Has your sleep been interrupted because of problems with your jaw or face?
0 1 2 3 4

31. Have you been upset because of problems with your jaw or face?
0 1 2 3 4

0 = never **1** = hardly ever **2** = sometimes **3** = fairly often **4** = very often

---

32. Have you found it difficult to relax because of problems with your jaw or face? **0 1 2 3 4**

33. Have you felt depressed because of problems with your jaw or face? **0 1 2 3 4**

34. Has your concentration been affected because of problems with your jaw or face? **0 1 2 3 4**

35. Have you been a bit embarrassed because of problems with your jaw or face? **0 1 2 3 4**

36. Have you avoided going out because of problems with your jaw or face? **0 1 2 3 4**

37. Have you been less tolerant of your spouse or family because of problems with your jaw or face? **0 1 2 3 4**

38. Have you had trouble getting on with other people because of problems with your jaw or face? **0 1 2 3 4**

39. Have you been a bit irritable because of problems with your jaw or face? **0 1 2 3 4**

40. Have you had difficulty doing your usual jobs because of problems with your jaw or face? **0 1 2 3 4**

41. Have you felt that your general health has worsened because of problems with your jaw or face? **0 1 2 3 4**

42. Have you suffered any financial loss because of problems with your jaw or face? **0 1 2 3 4**

43. Have you been unable to enjoy other people's company as much because of problems with your jaw or face? **0 1 2 3 4**

44. Have you felt that life in general was less satisfying because of problems with your jaw or face? **0 1 2 3 4**

45. Have you been totally unable to function because of problems with your jaw or face? **0 1 2 3 4**

46. Have you been unable to work to your full capacity because of problems with your jaw or face? **0 1 2 3 4**

Are you taking any medication for the pain? YES [ ] NO [ ]

Do you take any other medications? YES [ ] NO [ ]

Please list the names of all medications you are taking, the dose and frequency with which you take them:

name of medication	**dose**	**frequency**
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

To what extent is your pain relieved (**please circle**):

0 = not at all **1** = a little **2** = a lot **3** = completely

1. by pain-killers?	**0**	**1**	**2**	**3**
2. by your other efforts at dealing with it?	**0**	**1**	**2**	**3**
3. by the dentist or doctor?	**0**	**1**	**2**	**3**

What information have you been given regarding the cause of your pain?

_____

_____

_____

_____

_____

Thank you for completing the questionnaire.
Now please return the questionnaire to the researcher.

Patient no.....

EXAMINATION

1. **PAIN** (self-report)
   None.....0
   Right.....1
   Left.....2
   Both.....3

2. **DURATION** .....months

3. **DEVIATION** (on opening)
   None.....0
   Present.....1

4. **OPENING**
   Unassisted.....mm.
   Assisted.....mm.
   Overbite.....mm.

5. **JOINT SOUNDS** on opening

	R	L
None	0	0
Click	1	1
Crepitus	2	2

6. **JOINT SOUNDS** on closing

	R	L
None	0	0
Click	1	1
Crepitus	2	2

7. **JOINT SOUNDS** on excursions

	R	L
Lateral: None	0	0
Click	1	1
Crepitus	2	2
Protrusive: None	0	0
Click	1	1
Crepitus	2	2

8 **PALPATION** (tender)

		Right	Left	Both
temporalis	0	1	2	3
masseter	0	1	2	3
p. digastric	0	1	2	3
a. digastric	0	1	2	3
TMJ	0	1	2	3

Appendix II.

(Patient Information Letter)

CONFIDENTIAL

February 1999

Dear Madam / Sir,

You are due for a consultation appointment in the near future regarding face or head pain. I wonder if you might be interested in taking part in the following research project at that time:

Stress, Emotions and Disability in Facial Pain and Headache

This study looks at your own beliefs regarding the cause and consequences of your symptoms, as well as your mood and stress levels. Previous research has shown that these factors influence how well patients are able to adjust to their pain.
The results of the study will be used to help produce treatment programmes, which will aim to improve patient adjustment and prevent the development of long-term problems.

You are invited to complete a 30-minute questionnaire and to have your jaw and scalp muscles examined. You will be asked about your general health and any medicines which you are taking. Any information you give will be treated with complete confidentiality.

You are under no obligation to take part in this study but your participation will be gratefully appreciated and extremely helpful. If you decide to take part, you may withdraw at any time without having to give a reason. Your decision whether or not to take part will not affect your treatment in any way.

There is no need to decide in advance of your appointment but please contact me on the above mobile number should you have any prior questions about the study. I look forward to seeing you on the clinic.

Yours sincerely,

Geir Madland
Research Fellow

QUESTIONNAIRE FOR SUFFERERS OF FACE-ACHE AND HEADACHE

Thank you for agreeing to take part in this study.

Please follow the instructions for each section and answer **all** the questions.

A. Please give the following information about yourself:

age: _____ years

sex: male / female

status: single / living with a partner / married / separated / divorced / widowed

occupation: _____

number of years in full-time education: _____

B. Please indicate how frequently you experience the following symptoms, in relation to your face-/head-ache, **by ticking the appropriate box.**

	All of the time	Frequently	Occasionally	Never
Pain on waking				
Pain on yawning				
Pain on eating				
Difficulty in eating certain foods				
Loss of flavour in food				
Embarrassment when eating				
Incorrect bite				
Pain on speaking				
Speech not properly understood				
Awkwardness when speaking				
Spontaneous pain during the day				
Clicking jaw				
Stiff or locking jaw				
Lop-sided jaw				
Changing shape of jaw				
Concern over facial appearance				
Popping in the ears				
Tooth clenching or grinding				
Toothache				
Headache that worsens with physical activity such as walking stairs				
Headache with nausea				
Headache with vomiting				
Headache bothered by light				
Headache bothered by sounds				
Neck ache				
Back ache				
Difficulty sleeping				

C. We are interested in your own personal views of how you now see your face-/head-ache. Please indicate how much you agree or disagree with the following statements about your pain **by ticking the appropriate box.**

	Strongly agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagree
A germ or virus caused my pain					
Diet played a major role in causing my pain					
Pollution of the environment caused my pain					
My pain is hereditary - it runs in my family					
My pain started just by chance					
Stress was a major factor in causing my pain					
My pain is largely due to my own behaviour					
Other people played a large role in causing my pain					
My pain was caused by poor dental care in the past					
My state of mind played a major part in causing my pain					
The symptoms of my pain change a great deal from day to day					
The symptoms of my pain are distressing to me					
The symptoms of my pain are puzzling to me					
Changing my diet will help to control my pain					
My pain will improve in time					
My pain comes and goes in cycles					
My pain will last a short time					
There is very little that can be done to improve my pain					
There is a lot which I can do to control my symptoms					
My pain has major consequences for my life					
My pain has become easier to live with					
My pain has not had much effect on my life					
My treatment will be effective in curing my pain					
Recovery from my pain is largely dependent on chance or fate					
What I do can determine whether my pain gets better or worse					
My condition has strongly affected the way I see myself as a person					

	Strongly agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagree
My pain will last for a long time					
The symptoms of my pain affect many parts of my body					
I am aware of my symptoms all the time					
The symptoms of my pain are constant					
My pain will be controlled by rest					
My pain will go away on its own					
My pain is likely to be permanent rather than temporary					
My pain requires long-term care					
My condition has serious economic and financial consequences					
My pain is disabling					
My pain has strongly affected the way others see me					
My pain will be controlled by physical exercise					
My pain will be controlled by reduced stress					
My pain is a serious condition					
My pain is so severe as to prohibit work and other activities					

D. We are interested in how you respond to face-/head-ache.
Please indicate how often you do each of the following, in response to your pain, by **ticking beneath the appropriate number**:

IN RESPONSE TO MY FACE-/HEAD-ACHE.....	**0 not at all**	**1**	**2**	**3**	**4**	**5**	**6 very often**
I try to feel distant from the pain, almost as if the pain was in somebody else's body							
I leave the house and do something, such as going to the cinema or shopping							
I try to do something pleasant							
I don't think of it as pain but rather as a dull or warm feeling							
I read							
I tell myself to be brave and carry on despite the pain							
I tell myself that I can overcome the pain							
I count numbers in my head or run a song through my mind							
I just think of it as some other sensation, such as numbness							
I play mental games with myself to keep my mind off the pain							

IN RESPONSE TO MY FACE-/HEAD-ACHE.....	0 not at all	1	2	3	4	5	6 very often
I try not to think of it as my body, but rather as something separate from me							
I don't think about the pain							
I tell myself it doesn't hurt							
I tell myself I can't let the pain stand in the way of what I have to do							
I don't pay any attention to the pain							
No matter how bad it gets, I know I can handle it							
I pretend it's not there							
I replay in my mind pleasant experiences in the past							
I think of people I enjoy doing things with							
I imagine that the pain is outside of my body							
I just go on as if nothing happened							
I see it as a challenge and don't let it bother me							
Although it hurts, I just keep on going							
I try to be around other people							
I ignore it							
I think of things I enjoy doing							
I do anything to get my mind off the pain							
I do something I enjoy, such as watching TV or listening to music							
I pretend it's not a part of me							
I do something active, like household chores or projects							
It's terrible and I feel it's never going to get any better							
It's awful and I feel that it overwhelms me							
I feel my life isn't worth living							
I know somebody will be here to help me and it will go away for a while							
I pray to God it won't last long							
I try to think years ahead, what everything will be like after I've gotten rid of the pain							
I have faith in doctors that someday there will be a cure for my pain							
I worry all the time about whether it will end							
I pray for the pain to stop							
I feel I can't stand it anymore							
I rely on my faith in God							
I feel like I can't go on							
I feel I have control over the pain							
I am able to decrease the pain							

E. Please answer this section with reference to the pain you have had **over the past month**.

If a word **does not** describe your pain, tick **'NONE'** next to that word.
If a word **does** describe your pain, indicate whether you experience this sensation to a **'MILD'**, **'MODERATE'** or **'SEVERE'** degree, by ticking next to the relevant word.
Please make sure you place a tick in one of the categories for each word.

	NONE	MILD	MODERATE	SEVERE
THROBBING/PULSATING				
SHOOTING				
STABBING				
SHARP				
CRAMPING				
GNAWING				
HOT/BURNING				
ACHING				
HEAVY				
TENDER				
SPLITTING				
TIRING/EXHAUSTING				
SICKENING				
FEARFUL				
PUNISHING/CRUEL				
PRESSING/TIGHTENING				

a) Draw a line through the scale below to indicate where your pain has been **over the past month** (e.g. ——/——), imagining that the line indicates the ladder going from no pain to the worst possible pain.

NO PAIN ———————————————————— **WORST POSSIBLE PAIN**

b) Tick next to **one** of the following words / groups of words to indicate how intense your pain has been **over the past month**.

NO PAIN .....
MILD .....
DISCOMFORTING .....
DISTRESSING .....
HORRIBLE .....
EXCRUCIATING .....

F. Read each item and place a tick in the box opposite the reply which comes closest to how you have been feeling **in the past few weeks.**
Don't take too long over your replies, we want your immediate reaction.
Tick only one box in each section.

I feel tense or 'wound up':
- Most of the time [ ]
- A lot of the time [ ]
- Time to time, occasionally [ ]
- Not at all [ ]

I feel as if I am slowed down:
- Nearly all the time [ ]
- Very often [ ]
- Sometimes [ ]
- Not at all [ ]

I still enjoy the things I used to enjoy:
- Definitely as much [ ]
- Not quite as much [ ]
- Only a little [ ]
- Hardly at all [ ]

I get a sort of frightened feeling like 'butterflies' in my stomach:
- Not at all [ ]
- Occasionally [ ]
- Quite often [ ]
- Very often [ ]

I get a sort of frightened feeling as if something awful is about to happen:
- Very definitely [ ]
- Yes, but not too badly [ ]
- A little, it doesn't worry me [ ]
- Not at all [ ]

I have lost interest in my appearance:
- Definitely [ ]
- I don't take as much care as I should [ ]
- I may not take quite as much care [ ]
- I take just as much care as ever [ ]

I can laugh and see the funny side of things:
- As much as I always could [ ]
- Not quite so much now [ ]
- Definitely not so much now [ ]
- Not at all [ ]

I feel restless as if I have to be on the move:
- Very much indeed [ ]
- Quite a lot [ ]
- Not very much [ ]
- Not at all [ ]

Worrying thoughts go through my mind:
- A great deal of the time [ ]
- A lot of the time [ ]
- From time to time, not too often [ ]
- Only occasionally [ ]

I look forward with enjoyment to things:
- As much as I ever did [ ]
- Rather less than I used to [ ]
- Definitely less than I used to [ ]
- Hardly at all [ ]

I feel cheerful:
- Not at all [ ]
- Not often [ ]
- Sometimes [ ]
- Most of the time [ ]

I get sudden feelings of panic:
- Very often indeed [ ]
- Quite often [ ]
- Not very often [ ]
- Not at all [ ]

I can sit at ease and feel relaxed:
- Definitely [ ]
- Usually [ ]
- Not often [ ]
- Not at all [ ]

I can enjoy a good book or the TV:
- Often [ ]
- Sometimes [ ]
- Not often [ ]
- Very seldom [ ]

G. The questions in this scale ask you about your feelings and thoughts **during the last month**. The best approach is to answer each question fairly quickly. In each case, indicate how often you felt or thought in a certain way **by ticking the appropriate box**:

OVER THE PAST MONTH, HOW OFTEN HAVE YOU....	Never	Almost never	Some-times	Fairly often	Very often
been upset because of something that happened unexpectedly?					
felt that you were unable to control the important things in your life?					
felt nervous and stressed?					
felt confident about your ability to handle your personal problems?					
felt that things were going your way?					
found that you could not cope with all the things you had to do?					
been unable to control irritations in your life?					
felt that you were on top of things?					
been angered because of things that happened that were outside your control?					
felt difficulties were piling up so high that you could not overcome them?					

H. In this section there are a number of statements which people use to describe their reactions when they feel angry or furious. Read each statement and indicate **with a tick** how often you behave like this.

WHEN ANGRY OR FURIOUS....	Almost never	Some-times	Often	Almost always
I control my temper				
I express my anger				
I keep things in				
I am patient with others				
I pout or sulk				
I withdraw from people				
I make sarcastic remarks to others				
I keep my cool				
I do things like slam doors				
I boil inside, but I don't show it				
I control my behaviour				
I argue with others				
I tend to harbour grudges that I don't tell anyone about				
I strike out at whatever infuriates me				
I can stop myself from losing my temper				
I am secretly quite critical of others				
I am angrier than I am willing to admit				
I calm down faster than most other people				
I say nasty things				
I try to be tolerant and understanding				
I'm irritated a great deal more than other people are aware				
I lose my temper				
If someone annoys me I'm apt to tell him or her how I feel				
I control my angry feelings				

I. Using the scale provided as a guide, indicate how much you agree or disagree with each of the following statements **with a tick.**

	Strongly disagree	Moderately disagree	Neither agree nor disagree	Moderately agree	Strongly agree
I am often confused about what emotion I am feeling.					
It is difficult for me to find the right words for my feelings.					
I have physical sensations that even doctors don't understand.					
I am able to describe my feelings easily.					
I prefer to analyse problems rather than just describe them.					
When I am upset, I don't know if I am sad, frightened, or angry.					
I am often puzzled by sensations in my body.					
I prefer to just let things happen rather than to understand why they turned out that way.					
I have feelings that I can't quite identify.					
Being in touch with emotions is essential.					
I find it hard to describe how I feel about people.					
People tell me to describe my feelings more.					
I don't know what's going on inside me.					
I often don't know why I'm angry.					
I prefer talking to people about their daily activities rather than their feelings.					
I prefer to watch " light" entertainment shows rather than psychological dramas.					
It is difficult for me to reveal my innermost feelings, even to close friends.					
I can feel close to someone, even in moments of silence.					
I find examination of my feelings useful in solving emotional problems.					
Looking for hidden meanings in films or plays distracts from their enjoyment.					

J. Are you taking any medication for the pain? YES [ ] NO [ ]

Do you take any other medications? YES [ ] NO [ ]

Please list the names of all medications you are taking, the dose and frequency with which you take them:

name of medication	**dose**	**frequency**

K. Please answer this section with reference to the pain you have **right at this moment**.

a) Draw a line through the scale below (e.g. ——/——), imagining that the line indicates the ladder going from no pain to the worst possible pain.

NO PAIN ———————————————— **WORST POSSIBLE PAIN**

b) Tick next to **one** of the following words / groups of words to indicate how intense your pain is **right at this moment**.

NO PAIN .....
MILD .....
DISCOMFORTING .....
DISTRESSING .....
HORRIBLE .....
EXCRUCIATING .....

Thank you for completing the questionnaire, please return it to the researcher.

L. This section is to be completed by the researcher.

Patient no.....

Referral Centre: .....

Reason for referral: **FACE** or **HEAD**

		Face	Head
1.	**PAIN** (self-report)	Right.....1	1
		Left.....2	2
		Both.....3	3

2. **DURATION** .....months

FREQUENCY .....x/month (? < 15 < ?)

3. **DEVIATION** (on jaw opening) None.....0
Present.....1

4. **OPENING** Unassisted.....mm.
Assisted.....mm.
Overbite.....mm.

5. **JOINT SOUNDS** on opening	None.....R.....0	L.....0
	Click.....1	1
	Crepitus.....2	2

6. **JOINT SOUNDS** on closing	None.....R.....0	L.....0
	Click.....1	1
	Crepitus.....2	2

7. **JOINT SOUNDS** on excursions	Lateral: None.....R.....0	L.....0
	Click.....1	1
	Crepitus.....2	2
	Protrusive: None.....R.....0	L.....0
	Click.....1	1
	Crepitus.....2	2

8. **PALPATION** (tender)

		Right	Left	Both
pericranials	0	1	2	3
temporalis	0	1	2	3
masseter	0	1	2	3
subauricular	0	1	2	3
submandibular	0	1	2	3
TMJ	0	1	2	3

Appendix III.

Department of Oral Medicine
Telephone: **020 7915 1004** (24 hours)

CONFIDENTIAL

Dear Madam / Sir,

You have been referred to this hospital regarding jaw pain and are currently on a waiting list. In the meantime, I wonder if you might be interested in taking part in the following research treatment project:

A Self - Management Programme for Jaw Pain

On attending, following an assessment, a diagnosis of your pain condition will be made. If appropriate, you will be invited to participate in the project and to complete a 30-minute questionnaire. Any information you give will be treated with complete confidentiality.

You will then be randomly presented with one of two programmes on an easy-to-use computer, each lasting 10 minutes, and also asked to complete a second short questionnaire. We will then compare the effectiveness of the two programmes with two further questionnaires, one sent to you in the post six weeks later, and another at your final visit six months later.

You are under no obligation to take part in this study but your participation will be gratefully appreciated and extremely helpful. If you decide to take part, you may withdraw at any time without having to give a reason. If you decide not to take part, you will receive standard treatment instead.

Please contact me on the above number to arrange an appointment or to further discuss the study. If you get the answer machine, please leave a daytime telephone number for me to get back to you.

Please let me know one way or another within the next few days.

Yours faithfully,

Geir Madland
Clinical Tutor

CONFIDENTIAL

QUESTIONNAIRE 1

Thank you for agreeing to take part in this study.

Please follow the instructions for each section and answer **all** the questions.

A. Please give the following information about yourself:

age: _____ years

sex: male / female

status: single / living with a partner / married / separated / divorced / widowed

occupation: _____

number of years in full-time education (primary, secondary, higher, further): ___

B. We are interested in how **your jaw pain** affects you.
Please answer the following questions by **ticking** one box for each:

how often over the past month, have you.....	Never	Hardly ever	Some-times	Fairly often	Very often
had trouble pronouncing any words?					
felt that your sense of taste has worsened?					
had painful aching in your mouth?					
found it uncomfortable to eat any foods?					
been self-conscious?					
felt tense?					
found your diet unsatisfactory?					
had to interrupt meals?					
found it difficult to relax?					
been a bit embarrassed?					
been a bit irritable?					
had difficulty doing your usual jobs?					
felt that life in general was less satisfying?					
been totally unable to function?					

C. We are interested in your own personal views of how you now see your jaw pain. Please indicate how much you agree or disagree with the following statements about your pain **by ticking the appropriate box.**

	Strongly agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagre
A germ or virus caused my pain					
Diet played a major role in causing my pain					
Pollution of the environment caused my pain					
My pain is hereditary - it runs in my family					
My pain started just by chance					
Stress was a major factor in causing my pain					
My pain is largely due to my own behaviour					
Other people played a large role in causing my pain					
My pain was caused by poor dental care in the past					
My state of mind played a major part in causing my pain					
The symptoms of my pain change a great deal from day to day					
The symptoms of my pain are distressing to me					
The symptoms of my pain are puzzling to me					
Changing my diet will help to control my pain					
My pain will improve in time					
My pain comes and goes in cycles					
My pain will last a short time					
There is very little that can be done to improve my pain					
There is a lot which I can do to control my symptoms					
My pain has major consequences for my life					
My pain has become easier to live with					
My pain has not had much effect on my life					
My treatment will be effective in curing my pain					
Recovery from my pain is largely dependent on chance or fate					
What I do can determine whether my pain gets better or worse					
My condition has strongly affected the way I see myself as a person					
My pain will last for a long time					
The symptoms of my pain affect many parts of my body					
I am aware of my symptoms all the time					
The symptoms of my pain are constant					
My pain will be controlled by rest					
My pain will go away on its own					
My pain is likely to be permanent rather than temporary					
My pain requires long-term care					
My condition has serious economic and financial consequences					
My pain is disabling					
My pain has strongly affected the way others see me					
My pain will be controlled by physical exercise					
My pain will be controlled by reduced stress					
My pain is a serious condition					
My pain is so severe as to prohibit work and other activities					

D. We are interested in how you respond to jaw pain. Please indicate how often you do each of the following, in response to your pain, by **ticking beneath the appropriate number**:

IN RESPONSE TO MY JAW PAIN....	0 not at all	1	2	3	4	5	6 v o
I try to feel distant from the pain, almost as if the pain was in somebody else's body							
I leave the house and do something, such as going to the cinema or shopping							
I try to do something pleasant							
I don't think of it as pain but rather as a dull or warm feeling							
I read							
I tell myself to be brave and carry on despite the pain							
I tell myself that I can overcome the pain							
I count numbers in my head or run a song through my mind							
I just think of it as some other sensation, such as numbness							
I play mental games with myself to keep my mind off the pain							
I try not to think of it as my body, but rather as something separate from me							
I don't think about the pain							
I tell myself it doesn't hurt							
I tell myself I can't let the pain stand in the way of what I have to do							
I don't pay any attention to the pain							
No matter how bad it gets, I know I can handle it							
I pretend it's not there							
I replay in my mind pleasant experiences in the past							
I think of people I enjoy doing things with							
I imagine that the pain is outside of my body							
I just go on as if nothing happened							
I see it as a challenge and don't let it bother me							
Although it hurts, I just keep on going							
I try to be around other people							
I ignore it							
I think of things I enjoy doing							
I do anything to get my mind off the pain							
I do something I enjoy, such as watching TV or listening to music							
I pretend it's not a part of me							
I do something active, like household chores or projects							
It's terrible and I feel it's never going to get any better							
It's awful and I feel that it overwhelms me							
I feel my life isn't worth living							
I know somebody will be here to help me and it will go away for a while							
I pray to God it won't last long							
I try to think years ahead, what everything will be like after I've gotten rid of the pain							
I have faith in doctors that someday there will be a cure for my pain							
I worry all the time about whether it will end							
I pray for the pain to stop							
I feel I can't stand it anymore							
I rely on my faith in God							
I feel like I can't go on							
I feel I have control over the pain							
I am able to decrease the pain							

E. Please answer this section with reference to the pain you have had **over the past month.**
If a word **does not** describe your pain, tick **'NONE'** next to that word.
If a word **does** describe your pain, indicate whether you experience this sensation to a **'MILD'**, **'MODERATE'** or **'SEVERE'** degree, by ticking next to the relevant word.
Please make sure you place a tick in one of the categories for each word.

	NONE	MILD	MODERATE	SEVERE
THROBBING / PULSATING				
SHOOTING				
STABBING				
SHARP				
CRAMPING				
GNAWING				
HOT / BURNING				
ACHING				
HEAVY				
TENDER				
SPLITTING				
TIRING / EXHAUSTING				
SICKENING				
FEARFUL				
PUNISHING/CRUEL				

a) Draw a line through the scale below to indicate where your pain has been **over the past month** (e.g. ----/----), imagining that the line indicates the ladder going from no pain to the worst possible pain.

NO PAIN ---------- **WORST POSSIBLE PAIN**

b) Tick next to **one** of the following words / groups of words to indicate how intense your pain has been **over the past month.**

NO PAIN .....
MILD .....
DISCOMFORTING .....
DISTRESSING .....
HORRIBLE .....
EXCRUCIATING .....

F. This is a questionnaire designed to determine the way in which different people view certain important health-related issues.
Please answer these items carefully but **do not spend too long on any one item**. It is important that you respond according to your actual beliefs and not according to how you feel you should believe or how you think we want you to believe.

	strongly disagree	moder-ately disagree	slightly disagree	slightly agree	moder-ately agree	strongly agree
If I get sick, it is my own behaviour which determines how soon I get well again.						
No matter what I do, if I am going to get sick, I will get sick.						
Having regular contact with my doctor is the best way for me to avoid illness.						
Most things that affect my health happen to me by accident.						
Whenever I don't feel well, I should consult a medically trained professional.						
I am in control of my health.						
My famiily has a lot to do with my becoming sick or staying healthy.						
When I get sick, I am to blame.						
Luck plays a big part in determining how soon I will recover from an illness.						
Health professionals control my health.						
My good health is largely a matter of good fortune.						
The main thing which affects my health is what I myself do.						
If I take care of myself, I can avoid illness.						
When I recover from an illness, it's usually because other people (for example, doctors, nurses, family, friends) have been taking good care of me.						
No matter what I do, I'm likely to get sick.						
If it's meant to be, I will stay healthy.						
If I take the right actions, I can stay healthy.						
Regarding my health, I can only do what my doctor tells me to do.						

G. These questions are about your general confidence in yourself:

	not at all	barely true	moderately true	exactly true
I can always manage to solve difficult problems if I try hard enough.				
If someone opposes me, I can find means and ways to get what I want.				
It is easy for me to stick to my aims and accomplish my goals.				
I am confident that I could deal efficiently with unexpected events.				
Thanks to my resourcefulness, I know how to handle unforeseen situations.				
I can solve most problems if I invest the necessary effort.				
I can remain calm when facing difficulties because I can rely on my coping abilities.				
When I am confronted with a problem, I can usually find several solutions.				
If I am in a bind, I can usually think of something to do.				
No matter what comes my way, I'm usually able to handle it.				

H. A number of statements which people use to describe themselves are given below. Read each statement carefully, and then indicate, by placing a tick in the appropriate box, how you feel **right now,** that is **at this moment**.
There are no right or wrong answers. Do not spend too much time on any one statement.

	not at all	somewhat	moderately	very much
I feel calm				
I am tense				
I feel upset				
I am relaxed				
I feel content				
I am worried				

I. This section consists of 21 groups of statements. After reading each group carefully, **circle the number** next to the one statement in each group which best describes the way you have been feeling **the past week including today**. If several statements within a group seem to apply equally well, circle the highest number. Be sure that you do not choose more than one statement for any group.

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the tihings I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel paticularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticise or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticise myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry any more than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleep Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable then usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18 Changes of Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.

2a My appetite is much less than before.
2b My appetite is much greater than usual.

3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

J. Whom have you seen regarding your face pain **over the past 6 months**, and how often?

Dentist	.....times
Doctor	.....times
Hospital Specialist (please specify)	.....times
Other (please specify)	.....times

Are you taking any medication for the pain? YES [ ] NO [ ]

Do you take any other medications? YES [ ] NO [ ]

Please list the names of all medications you are taking, the dose and frequency with which you take them:

name of medication	**dose**	**frequency**

Have you had any other forms of treatment (please specify) **?**

.....

.....

Thank you. Please let the researcher know you have finished.

This section is to be completed by the researcher. Patient no.....

Referral Centre.....

1. **PAIN** (self-report)
   Right.....1
   Left.....2
   Both.....3

2. **DURATION** .....months

   FREQUENCY .....x/month (? < 15 < ?)

3. **DEVIATION** (on jaw opening)
   None.....0
   Present.....1

4. **OPENING**
   Unassisted.....mm.
   Assisted.....mm.
   Overbite.....mm.

5. **JOINT SOUNDS** on opening

None	R.....0	L.....0
Click	1	1
Crepitus	2	2

6. **JOINT SOUNDS** on closing

None	R.....0	L.....0
Click	1	1
Crepitus	2	2

7. **JOINT SOUNDS** on excursions

Lateral:	None	R.....0	L.....0
	Click	1	1
	Crepitus	2	2
Protrusive:	None	R.....0	L.....0
	Click	1	1
	Crepitus	2	2

8. **PALPATION** (tender)

		Right	Left	Both
pericranials	0	1	2	3
temporalis	0	1	2	3
masseter	0	1	2	3
subauricular	0	1	2	3
submandibular	0	1	2	3
TMJ	0	1	2	3

Questionnaire 2 **To be completed after the programme.**

We would be grateful for your views on the programme you have just watched.

How useful was the programme (please tick)?

	1 bad	2	3	4	5 excellent
Overall					
Information					
Advice					

Was the programme the right length?

Yes	No, too short	No, too long

Which parts did you find most useful and why?.....

.....

Which parts did you find least useful and why?.....

.....

What do you feel you have learnt from this programme?.....

.....

How do you rate this programme in relation to:

	1 bad	2	3	4	5 excellent
a written leaflet?					
a personal consultation?					

Please complete the following by referring to any aspect of the programme:

The best thing about the programme was.....

.....

Another good thing was.....

.....

The worst thing about the programme was.....

.....

Another bad thing was.....

.....

I would have liked more.....

.....

I would have liked less.....

.....

How likely are you now to practise any suggested self-help exercises?

very likely	quite likely	don't know	quite unlikely	very unlikely

Would you have liked a copy of the programme to take home with you? YES NO

Thank you. Please return the whole questionnaire to the researcher.

Questionnaire 3

*In addition to sections **B.**, **E.**, **H.** and **I.** from Questionnaire 1:*

Please indicate whether you agree with each of the following statements about yourself, by ticking the appropriate box.

	Strongly agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagree
I have tried everything that people have recommended to manage my pain and nothing helps.					
My pain is a medical problem and I should be dealing with physicians about it.					
Everybody I speak with tells me that I have to learn to live with my pain, but I don't see why I should have to.					
I still think despite what doctors tell me, there must be some surgical procedure or medication that would get rid of my pain.					
The best thing I can do is find a doctor who can figure out how to get rid of my pain once and for all.					
Why can't someone just do something to take away my pain?					
All of this talk about how to cope better is a waste of time.					
I have been thinking that the way I cope with my pain could improve.					
I have recently realised that there is no medical cure for my pain condition, so I want to learn some ways to cope with it.					
Even if my pain doesn't go away, I am ready to start changing how I deal with it.					
I realise now that it's time to come up with a better plan to cope with my pain problem.					
I am beginning to wonder if I need to get some help to develop skills for dealing with my pain.					
I have recently figured out that it's up to me to deal better with my pain.					
I have recently come to the conclusion that it's time for me to change how I cope with my pain.					

	Strongly agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagree
I'm starting to wonder whether it's up to me to manage my pain rather than relying on physicians.					
I have been thinking that doctors can only help so much in managing my pain and that the rest is up to me.					
I have been wondering if there is some thing I could do to manage my pain better.					
I am developing new ways to cope with my pain.					
I have started to come up with strategies to help myself control my pain.					
I'm getting help learning some strategies for coping better with my pain.					
I am learning to help myself control my pain without doctors.					
I am testing out some coping skills to manage my pain better.					
I am learning ways to control my pain other than with medications or surgery.					
I have learned some good ways to keep my pain problem from interfering with my life.					
When my pain flares up, I find myself automatically using coping strategies that have worked in the past, such as a relaxation exercise or mental distraction technique.					
I am using some strategies that help me better deal with my pain problem on a day-to-day basis.					
I use what I have learnt to help keep my pain under control.					
I am currently using some suggestions people have made about how to live with my pain problem.					
I have incorporated strategies for dealing with my pain into my everyday life.					
I have made a lot of progress in coping with my pain.					